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## The urethral support system during pregnancy and after childbirth

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# STELLINGEN

behorend bij het proefschrift van Jacobus Wijma

**The urethral support system in pregnancy and after childbirth**

Groningen, 10 december 2007

1. De toename van de rusthoek van de blaashals ten opzichte van de symfyse wordt reeds in de 12<sup>e</sup> zwangerschapsweek vastgesteld en pleit daarom meer voor een hormonale dan voor een mechanische beïnvloeding van het support system. (Hoofdstuk 2)
2. Het feit dat de prevalentie van incontinentie postpartum afneemt ten opzichte van antepartum, terwijl de mobiliteit toeneemt, bewijst dat hormonale veranderingen van grotere invloed zijn dan veranderde mobiliteit. (Hoofdstuk 3)
3. De verandering van de mobiliteit van de blaashals na een vaginale baring wordt meer bepaald door intrinsieke eigenschappen (genetisch) dan door extrinsieke invloeden (partus). (Hoofdstuk 4)
4. De mobiliteit van de blaashals is een optelsom van lineaire en niet-lineaire processen en behoeft daarom een nadere beschrijving. (Hoofdstuk 5)
5. Voor een geslaagde vaginale baring is adaptatie van het baringskanaal noodzakelijk en aangetoond. (Hoofdstuk 1 en 5)
6. Luiertesten voor incontinentie meten vochtverlies over een bepaalde periode maar zijn niet bruikbaar om door patiënten ingevulde vragenlijsten over urineverlies te vervangen. (Hoofdstuk 6)
7. De correlatie tussen de afname van het aantal publicaties over hormonale substitutietherapie en de omzet in die markt, doet vermoeden dat het toenemend aantal publicaties over bekkenbodempromotie iets zegt over een groeiende omzet in deze markt.
8. Omdat bij vrouwen van 65 jaar en ouder de leeftijd een betere voorspeller voor urine- incontinentie is dan de pariteit, zal het electief verrichten van een keizersnede het probleem van incontinentie op latere leeftijd niet oplossen.
9. Zolang de beste stuurder aan wal blijven staan, kan men op zee zijn eigen koers varen.
10. Door het opheffen van de gelijkgerichte financiering van de medisch specialist en het ziekenhuis is het geïntegreerd medisch specialistisch bedrijf per 1 januari 2008 gedisintegreerd.

11. Bij een identiek uurtarief voor alle medisch specialisten in Nederland is een significant omzetverschil zonder significant inzetverschil uitsluitend te verklaren doordat bij het ene specialisme meer uren in een uur gaan dan bij het andere.
12. Het feit dat de vervenersboerderij aan de Hoornsedijk 13 te Haren is benoemd tot rijksmonument zegt meer over het rijk dan over het monument.
13. Terwijl de patiënt op zoek is naar een sensitieve dokter, is de dokter op zoek naar een specifieke test.



# **THE URETHRAL SUPPORT SYSTEM DURING PREGNANCY AND AFTER CHILDBIRTH**

J Wijma

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RIJKSUNIVERSITEIT GRONINGEN

# THE URETHRAL SUPPORT SYSTEM IN PREGNANCY AND AFTER CHILDBIRTH

## Proefschrift

ter verkrijging van het doctoraat in de  
Medische Wetenschappen  
aan de Rijksuniversiteit Groningen  
op gezag van de  
Rector Magnificus, dr. F. Zwarts,  
in het openbaar te verdedigen op  
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om 14.45 uur

door

**Jacobus Wijma**

geboren op 8 augustus 1958  
te Leeuwarden



Promotor: Prof.dr. J.G. Aarnoudse

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Prof.dr. M.E. Vierhout  
Prof.dr. A.G.J. van der Zee

*“onze huidige tevredenheid weerspiegelt misschien eerder het gebrek aan gegevens dan de voortreffelijkheid van de theorie”*

Sir Martin Rees (1942), astronoom en astrofysicus

Aan

mijn vader Geert Wijma (14 april 1926 – 14 oktober 2006)

mijn moeder Maria Golida Wijma-Post (5 augustus 1928 – 1 september 2007)



## CONTENTS

<b>1</b>	Introduction	9
<b>2</b>	Anatomical and functional changes in the lower urinary tract during pregnancy <i>Wijma J, Weis Potters AE, de Wolf BT, Tinga DJ and Aarnoudse JG (2001) Anatomical and functional changes in the lower urinary tract during pregnancy. BJOG 108:726-32.</i>	21
<b>3</b>	Anatomical and functional changes in the lower urinary tract following spontaneous vaginal delivery <i>Wijma J, Potters AE, de Wolf BT, Tinga DJ and Aarnoudse JG (2003) Anatomical and functional changes in the lower urinary tract following spontaneous vaginal delivery. BJOG 108:726-32.</i>	35
<b>4</b>	Pelvic floor characteristics after spontaneous and operative vaginal delivery <i>Serial studies prior to labor up to six months postpartum.</i>	47
<b>5</b>	Displacement and recovery of the vesical neck position during pregnancy and after childbirth <i>Wijma J, Weis Potters AE, van der Mark TW, Tinga DJ and Aarnoudse JG (2007) Displacement and recovery of the vesical neck position during pregnancy and after childbirth. Neurourol Urodyn 26:372-6.</i>	59
<b>6</b>	The diagnostic strength of the 24-h pad test for self-reported symptoms of urinary incontinence in pregnancy and after childbirth <i>Wijma J, Weis Potters AE, Tinga DJ and Aarnoudse JG. The diagnostic strength of the 24-h pad test for self-reported symptoms of urinary incontinence in pregnancy and after childbirth. Int Urogynecol J Pelvic Floor Dysfunct 2007 Oct 10; [Epub ahead of print]</i>	75
<b>7</b>	Summary and conclusions	87

<b>8</b>	Samenvatting en conclusies (Summary and conclusions in Dutch)	99
	Nawoord	107
	Curriculum vitae	108



PS.

# CHAPTER 1

## Introduction

## 1.1 INCONTINENCE IN PREGNANCY AND AFTER CHILDBIRTH

Pelvic floor dysfunction in women is a major health problem. Symptoms are protrusion of vaginal tissue, voiding difficulties, urinary incontinence, stool problems and sexual dysfunction. Many of these women may eventually require surgery for pelvic floor dysfunction, especially for prolaps and urinary incontinence. This thesis focuses on urinary incontinence and especially on the etiological aspects of pregnancy and childbirth. In a community survey MacLennan reported a prevalence of all types of self-reported urinary incontinence in women is 35.3%. Urinary incontinence increased after pregnancy according to parity and age. The highest prevalence (51.9%) is reported in women aged 70-74 years<sup>1-4</sup>. Numerous epidemiological studies have associated pelvic floor dysfunction with prior vaginal delivery<sup>1;5-15</sup>. In contrary there are only a very few prospective studies on the effect of pregnancy and childbirth on the urethral support system<sup>16-18</sup>, nowadays considered as the important issue in urinary control<sup>19</sup>.

It is generally accepted that as pregnancy proceeds a growing number of women complains of urinary incontinence. Accidental loss of urine is reported in 17-25% of women in early pregnancy<sup>20</sup> and in up to 36-67% in late pregnancy<sup>21</sup>. Moreover, the degree of incontinence worsens as pregnancy advances<sup>21</sup>. Urinary incontinence during pregnancy is ascribed to detrusor instability resulting from changes in hormone levels<sup>22</sup> and to anatomical changes due to the growing uterus and the engagement of the fetal head in the pelvis<sup>23</sup>. Others<sup>24</sup> have suggested that reduced fascial strength and subsequent weakness of the pelvic supports might contribute to urinary incontinence during pregnancy.

Until now, it is not clear to what extent pregnancy itself or vaginal delivery contributes to the development of urinary incontinence in later life. Following vaginal delivery neuromuscular damage and bladder neck hypermobility, indicating a change in pelvic floor function, has been confirmed<sup>25;26</sup>. Nevertheless in the great majority of women the incontinence has disappeared six months after delivery<sup>13;27</sup>.

## 1.2 THE URINARY CONTINENCE CONTROL SYSTEM

The control system for urinary continence is a complex network with several components. Anatomical it consist of the urethral support system and sphincteric

closing system<sup>19</sup>.

The sphincteric closing system consists of urethral striated muscles, the urethral smooth muscles, and the vascular elements within the submucosa<sup>28</sup>. The urethral muscles maintain continence in various ways. The striated muscles are well-suited to maintain constant tone as well as allow voluntary increase in tone to provide additional continence protection<sup>29</sup>. Moreover striated muscles are capable to compress the lumen. Smooth muscles may also play a role by surrounding the inner vascular layer, suggesting a role in constricting the lumen.

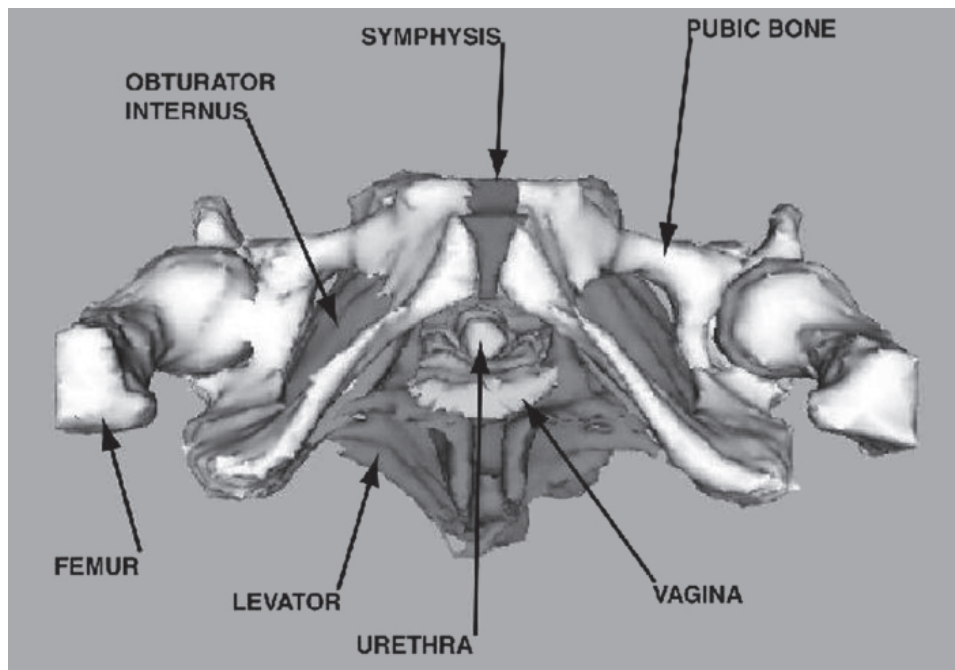
This sphincteric closing system yields a closing pressure which may deteriorate due to age<sup>30</sup> and neurological injury<sup>31;32</sup>. Studies on the effect of vaginal birth on the sphincter mechanism reveal decreases in urethral closure pressure as a result of vaginal birth<sup>17;33</sup>.

The urethral support system consists of all the structures extrinsic to the urethra that provide a supportive layer upon which the urethra rests<sup>34</sup>. The following structures can be distinguished:

1. The connective tissue sheath covering the ventral aspects of the urethra and the rhabdosphincter, which may be called ventral urethral fascia, which connects the right and left fasciae of the levator. Contraction of the levator narrows the pre urethral space and an ascending movement of the urethra and the rhabdosphincter.
2. The fasciae of the levator ani muscle, especially the right and left tendinous arch.
3. The strong dorsal structure of the urethra and the rhabdosphincter to the ventral wall of the vagina.

The m. pubo rectalis and the m. pubo coccygeus form a U shape as they originate from the pubic bone on either side of the midline and pass behind the rectum to form a sling. Composed of type 1 striated muscle suited to maintain constant tone<sup>35</sup>. It is this constant tone that keeps the urogenital hiatus closed<sup>36</sup>. The ileococcygeus muscles arise laterally from the arcus tendineus levator ani and form a horizontal sheet that spans the opening in the posterior region of the pelvis, thereby providing a shelf upon which the pelvic organs rest. In this respect the biomechanical properties of the pelvic floor are of great importance, since the pelvic floor must be capable in providing sufficient back pressure for urethral closure.





*With permission from: Hoyte L. Two- and 3-dimensional MRI comparison of levator ani structure, volume, and integrity in women with stress incontinence and prolapse. American journal of obstetrics and gynecology 2001; 185(1):11-19.*

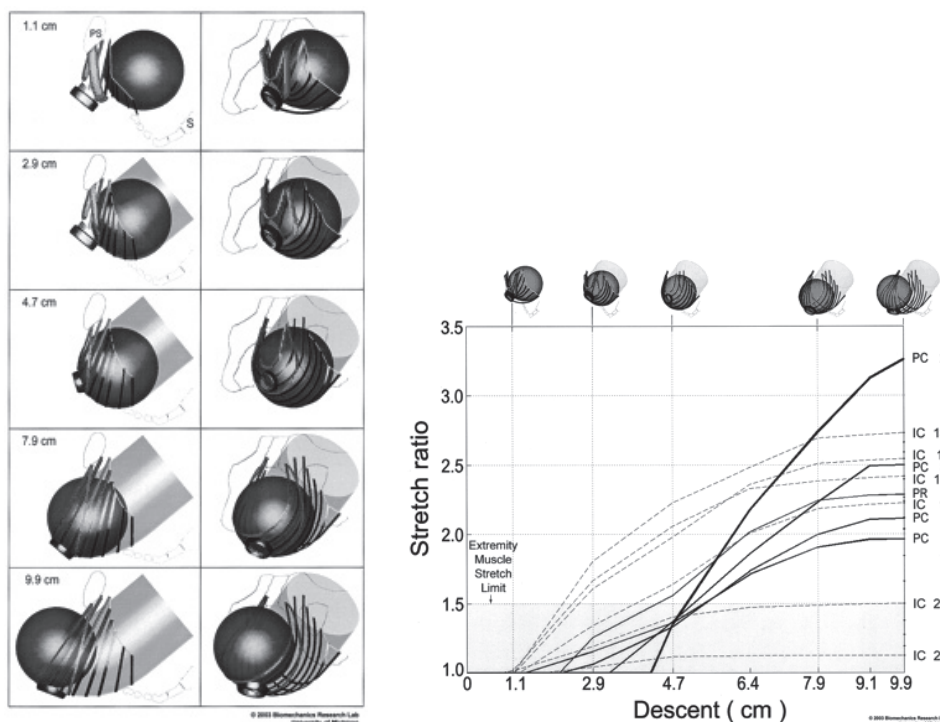
In a hard cough intra abdominal pressure can increase by about 150 cm H<sub>2</sub>O. Ultrasound studies have shown that during coughing the inferior abdominal contents are forced to move caudo dorsally (downwards), presumably due to a simultaneous contraction of the diaphragm and abdominal wall muscles. The proximal urethra and the anterior vaginal wall are intimately connected and attached to the muscles of the pelvic diaphragm and to the arcus tendineus fasciae pelvis. Support of the urethra at rest comes from both its attachments to the arcus tendineus fasciae pelvis and the resting tone of the muscles of the pelvic diaphragm<sup>37</sup>. Therefore, the mobility of the urethro-vesical junction can be used as an index of the mobility of these pelvic floor structures. The downward motion of the bladder neck visible in the ultrasound picture means that its surrounding tissues acquire downward momentum. This downward momentum must then be arrested by stretch resistance of the pelvic floor structures. As the downward momentum of the abdominal contents is slowed by the stretch of the pelvic floor structures, this movement compresses the proximal intra abdominal portion of the urethra against the underlying supportive structures, which is composed of the endopelvic fasciae, the vagina, and the levator ani muscles<sup>19</sup>.

### 1.3 DEFINITION OF THE PROBLEM

#### *Overstretching of the hiatus urogenitalis during pregnancy and after childbirth*

The ventral fasciae and the fasciae of the levator ani may be separated from the anterior rhabdosphincter. Also, overstretching of the vaginal wall may lead to disruption of the dorsal urethra and rhabdosphincter from the vaginal wall.

Birth imposes a remarkable degree of distension in the muscles and fasciae. If one assumes the diameter of the moulded fetal head (9cm, based on the data from Chitty et al<sup>38</sup>) is approximately 4 times the initial diameter of the urogenital hiatus (2.5 cm, according to the data of DeLancey and Hurd<sup>36</sup>) in the levator ani muscles through which the fetal head must pass during the second stage of labour. The theoretically needed stretch ratio is about  $9/2.5 = 3.6$ . In a computerized model of the passing fetal head through the hiatus urogenitalis Lien concluded that a maximal stretch ratio of 3.26 was found in the pubococcygeal muscles<sup>39</sup>. This is 217% of the maximum stretch as defined for non gravid muscles, 1.5.



With permission from: Lien KC, Mooney B, DeLancey JO, Ashton-Miller JA. Levator ani muscle stretch induced by simulated vaginal birth. *Obstet Gynecol* 2004; 103(1):31-40.

When damage has occurred in the levator ani muscle and or fasciae the anatomical defect is noted as a tendency towards increased size of the urogenital hiatus and vertical elongation of the levator plate<sup>40</sup>. The increased area of the urogenital hiatus is secondary more to an increase in anterior posterior diameter and less to an increase in transverse diameter<sup>36,41</sup>. When the ileococcygeus muscle is damaged, the resulting anatomical defect is a tendency towards vertical elongation of the levator plate<sup>42</sup>. This downward sagging of the levator plate increases the size of the urogenital hiatus<sup>36</sup> and results in loss of the valve mechanism that normally prevent pelvic organ prolaps.

*As the majority of women has no symptomatic pelvic organ prolaps after vaginal delivery we postulate that both fascia and muscles must have a remarkable accommodation of the level of tissue stretch needed for vaginal birth without major injury of pelvic floor.* During childbirth the urogenital hiatus has to adapt to the passing foetus in a limited time. The increase of the perimeter and/or the straining capacities of the tissue (compliance) of the hiatus urogenitalis might facilitate this process.

*There is great scarcity of prospective studies on urinary incontinence during pregnancy and after childbirth.* The wide variation of the reported prevalence and the uncertainty concerning the changes in the continence control system, prompted us to investigate these issues in a prospective longitudinal study in a homogeneous cohort of nulliparous pregnant women. Dynamic serial perineal ultrasound measurements of pelvic floor characteristics may give us a better understanding of the role and the changes in the continence control mechanism, both during pregnancy and after childbirth.

## 1.4 AIMS OF THE STUDY

*Assessment of the urethral support system during pregnancy and after childbirth, perineal ultrasonography, compliance and hysteresis*

By analysing the position and the movements of the urethra vesical junction (UVJ) in relation to the changes of the abdominal pressure one analyses the capacity and the stretch resistance of the urethral support system. Generally, tissue distensibility is determined not only by the elasticity of the constituent muscle and connective fibers itself but also by the geometrical arrangement of those fibers. Two parameters

are in use to describe the mechanical properties of tissue: Young's modulus, or its reciprocal (the compliance), to quantify the pure elastic behaviour of tissue in which the geometrical structure of the fibers remain unaltered, and hysteresis: the failure of tissue to follow the same course during relaxation as it did during distension. The latter parameter is thought to be the result of shifts in the geometrical structure of the fibers with respect to each other, and can be interpreted as a form of internal friction within the tissue. We used perineal ultrasound of the UVJ movements. It can be applied to get on line registration, and can be used freely in pregnancy without radiation, with limited discomfort and great availability. We had experience with the method since 1991, when we first reported on the use of ultrasound in the diagnosis of urinary incontinence<sup>43;44</sup>. By the use of perineal ultrasound and simultaneous abdominal pressure measurement we were able to measure the displacement and the recovery of the vesical neck in relation to the increase of abdominal pressure during coughing and during Valsalva manoeuvre. In general, deformation under load for tissue is seldomly linear. To quantify such deformation we decomposed deformation into elasticity, approximated by a linear model and hysteresis, estimated by a non linear model. These measurements were made throughout pregnancy and after childbirth resulting in serial measurement of the biomechanical properties of the vesical neck supporting structures during pregnancy and after childbirth.

#### *Assessment of functional changes of the pelvic floor*

(Functional) injury of pelvic floor, leading to changes of pelvic floor stretch resistance may lead to pelvic floor dysfunction, which includes urinary incontinence. Part of our study therefore was to measure the incidence of this symptom of pelvic floor dysfunction. Women completed questionnaires and visual analogue scales on symptoms of urinary incontinence, moreover 24-hour pad tests were used to objectify urine loss.

In chapter 2 we assessed the prevalence and the development of urinary incontinence in nulliparous pregnant women, both subjectively and objectively, and we investigated the relation of urinary incontinence with the mobility of the urethro-vesical junction measured by perineal ultrasound.

In chapter 3 we assessed the prevalence of urinary incontinence after spontaneous vaginal delivery and its relation with changes in the static and dynamic function of the pelvic floor.

In chapter 4 we compared women with spontaneous and operative vaginal delivery for urinary incontinence data and for pelvic floor characteristics

In chapter 5 we focused on displacement and recovery of the vesical neck position during pregnancy and after childbirth, especially we discriminated between compliance of the vesical neck supporting structures with and without pelvic floor contraction.

In chapter 6 we assessed the clinical usefulness of the 24-hour pad test in pregnancy and after childbirth in terms of the relationship between objective urine loss and the self reported symptoms of urinary incontinence.

Chapter 7 is the summary and conclusions and the Dutch summary is in Chapter 8.

## REFERENCE LIST

- (1) MacLennan AH, Taylor AW, Wilson DH, Wilson D. The prevalence of pelvic floor disorders and their relationship to gender, age, parity and mode of delivery. *BJOG* 2000; 107(12):1460-1470.
- (2) Peyrat L. Prevalence and risk factors of urinary incontinence in young and middle-aged women. *BJU international* 2002; 89(1):61-66.
- (3) Lukacz ES, Lawrence JM, Contreras R, Nager CW, Luber KM. Parity, mode of delivery, and pelvic floor disorders. *Obstet Gynecol* 2006; 107(6):1253-1260.
- (4) Chiarelli P, Brown WJ. Leaking urine in Australian women: prevalence and associated conditions. *Women Health* 1999; 29(1):1-13.
- (5) Demirci F. The effects of vaginal delivery and cesarean section on bladder neck mobility and stress urinary incontinence. *International urogynecology journal* 2001; 12(2):129-133.
- (6) Dolan LM. Stress incontinence and pelvic floor neurophysiology 15 years after the first delivery. *BJOG* 2003; 110(12):1107-1114.
- (7) Eason E. Effects of carrying a pregnancy and of method of delivery on urinary incontinence: a prospective cohort study. *BMC pregnancy and childbirth* 2004; 4(1):4.
- (8) Farrell SA, Allen VM, Baskett TF. Parturition and urinary incontinence in primiparas. *Obstet Gynecol* 2001; 97(3):350-356.

- (9) Meyer S, Hohlfield P, Achtari C, Russolo A, De Grandi P. Birth trauma: short and long term effects of forceps delivery compared with spontaneous delivery on various pelvic floor parameters. *BJOG* 2000; 107(11):1360-1365.
- (10) Rortveit G. Urinary incontinence after vaginal delivery or cesarean section. *The New England journal of medicine* 2003; 348(10):900-907.
- (11) Rortveit G, Daltveit AK, Hannestad YS, Hunskaar S. Vaginal delivery parameters and urinary incontinence: the Norwegian EPINCONT study. *Am J Obstet Gynecol* 2003; 189(5):1268-1274.
- (12) Snooks SJ, Swash M, Mathers SE, Henry MM. Effect of vaginal delivery on the pelvic floor: a 5-year follow-up. *Br J Surg* 1990; 77(12):1358-1360.
- (13) Viktrup L. The risk of stress incontinence 5 years after first delivery. *American journal of obstetrics and gynecology* 2001; 185(1):82-87.
- (14) Viktrup L, Lose G, Rolff M, Barfoed K. The symptom of stress incontinence caused by pregnancy or delivery in primiparas. *Obstet Gynecol* 1992; 79(6):945-949.
- (15) Willis S. Childbirth and incontinence: a prospective study on anal sphincter morphology and function before and early after vaginal delivery. *Langenbeck's archives of surgery* 2002; 387(2):101-107.
- (16) Dietz HP, Clarke B, Vancaillie TG. Vaginal childbirth and bladder neck mobility. *Aust N Z J Obstet Gynaecol* 2002; 42(5):522-525.
- (17) van Geelen JM, Lemmens WA, Eskes TK, Martin CB, Jr. The urethral pressure profile in pregnancy and after delivery in healthy nulliparous women. *Am J Obstet Gynecol* 1982; 144(6):636-649.
- (18) King JK, Freeman RM. Is antenatal bladder neck mobility a risk factor for postpartum stress incontinence? *Br J Obstet Gynaecol* 1998; 105(12):1300-1307.
- (19) Ashton-Miller JA, Howard D, DeLancey JO. The functional anatomy of the female pelvic floor and stress continence control system. *Scand J Urol Nephrol Suppl* 2001;(207):1-7.
- (20) Stanton SL, Kerr-Wilson R, Harris VG. The incidence of urological symptoms in normal pregnancy. *Br J Obstet Gynaecol* 1980; 87(10):897-900.
- (21) FRANCIS WJ. The onset of stress incontinence. *J Obstet Gynaecol Br Emp* 1960; 67:899-903.
- (22) Miodrag A, Castleden CM, Vallance TR. Sex hormones and the female urinary tract. *Drugs* 1988; 36(4):491-504.
- (23) Peschers U, Schaer G, Anthuber C, DeLancey JO, Schuessler B. Changes in vesical neck mobility following vaginal delivery. *Obstet Gynecol* 1996; 88(6):1001-1006.

- (24) Landon CR, Crofts CE, Smith ARB, Trowbridge EA. Mechanical properties of fascia during pregnancy: a possible factor in the development of stress incontinence of urine. *Contemp Rev Obstet Gynaecol* 1990; 2:40-46.
- (25) Sultan AH, Kamm MA, Hudson CN. Pudendal nerve damage during labour: prospective study before and after childbirth. *Br J Obstet Gynaecol* 1994; 101(1):22-28.
- (26) Meyer S, Schreyer A, De Grandi P, Hohlfeld P. The effects of birth on urinary continence mechanisms and other pelvic-floor characteristics. *Obstet Gynecol* 1998; 92(4 Pt 1):613-618.
- (27) Thorp JM, Jr., Norton PA, Wall LL, Kuller JA, Eucker B, Wells E. Urinary incontinence in pregnancy and the puerperium: a prospective study. *Am J Obstet Gynecol* 1999; 181(2):266-273.
- (28) Strohbehn K, Quint LE, Prince MR, Wojno KJ, DeLancey JO. Magnetic resonance imaging anatomy of the female urethra: a direct histologic comparison. *Obstet Gynecol* 1996; 88(5):750-756.
- (29) Gosling JA. A comparative study of the human external sphincter and periurethral levator ani muscles. *British journal of urology* 1981; 53(1):35-41.
- (30) Rud T, Andersson KE, Asmussen M, Hunting A, Ulmsten U. Factors maintaining the intraurethral pressure in women. *Invest Urol* 1980; 17(4):343-347.
- (31) Hilton P, Stanton SL. Urethral pressure measurement by microtransducer: the results in symptom-free women and in those with genuine stress incontinence. *Br J Obstet Gynaecol* 1983; 90(10):919-933.
- (32) Snooks SJ, Badenoch DF, Tiptaft RC, Swash M. Perineal nerve damage in genuine stress urinary incontinence. An electrophysiological study. *Br J Urol* 1985; 57(4):422-426.
- (33) Iosif S, Ulmsten U. Comparative urodynamic studies of continent and stress incontinent women in pregnancy and in the puerperium. *Am J Obstet Gynecol* 1981; 140(6):645-650.
- (34) Fritsch H, Pinggera GM, Lienemann A, Mitterberger M, Bartsch G, Strasser H. What are the supportive structures of the female urethra? *Neurourol Urodyn* 2006; 25(2):128-134.
- (35) Critchley HO, Dixon JS, Gosling JA. Comparative study of the periurethral and perianal parts of the human levator ani muscle. *Urol Int* 1980; 35(3):226-232.
- (36) DeLancey JO. Size of the urogenital hiatus in the levator ani muscles in normal women and women with pelvic organ prolapse. *Obstetrics and gynecology* 1998; 91(3):364-368.
- (37) DeLancey JO. Correlative study of paraurethral anatomy. *Obstet Gynecol* 1986; 68(1):91-97.

- (38) Chitty LS, Altman DG, Henderson A, Campbell S. Charts of fetal size: 2. Head measurements. *Br J Obstet Gynaecol* 1994; 101(1):35-43.
- (39) Lien KC, Mooney B, DeLancey JO, Ashton-Miller JA. Levator ani muscle stretch induced by simulated vaginal birth. *Obstet Gynecol* 2004; 103(1):31-40.
- (40) Huang WC, Yang SH, Yang JM. Anatomical and functional significance of urogenital hiatus in primary urodynamic stress incontinence. *Ultrasound Obstet Gynecol* 2006; 27(1):71-77.
- (41) Kearney R, Sawhney R, DeLancey JO. Levator ani muscle anatomy evaluated by origin-insertion pairs. *Obstet Gynecol* 2004; 104(1):168-173.
- (42) DeLancey JO. Anatomy and biomechanics of genital prolapse. *Clin Obstet Gynecol* 1993; 36(4):897-909.
- (43) Wijma J, Tinga DJ, Visser GH. Perineal ultrasonography in women with stress incontinence and controls: the role of the pelvic floor muscles. *Gynecol Obstet Invest* 1991; 32(3):176-179.
- (44) Wijma J, Tinga DJ, Visser GH. Compensatory mechanisms which prevent urinary incontinence in aging women. *Gynecol Obstet Invest* 1992; 33(2):102-104.







# CHAPTER 2

## Anatomical and functional changes in the lower urinary tract during pregnancy

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## ABSTRACT

**Objective** To assess the prevalence and the development of urinary incontinence in nulliparous pregnant women, both subjectively and objectively, and to investigate the relation of incontinence with the mobility of the urethro-vesical junction measured by perineal ultrasound.

**Design** A prospective longitudinal study.

**Setting** University Hospital and Martini Hospital Groningen, the Netherlands.

**Population** A cohort of 117 nulliparous pregnant women and 27 nulliparous non-pregnant controls.

**Methods** Urinary incontinence was measured by a questionnaire and by a 24-hour pad test. The position of the urethro-vesical junction and its mobility were measured by perineal ultrasound.

**Main outcome measure** Prevalence of urinary incontinence; mobility of the urethro-vesical junction, indicated by the displacement/pressure coefficient.

**Results** Up to 35% of the women reported urinary incontinence in pregnancy, and 20% of the women had a positive pad test. The angle of the urethro-vesical junction angle at rest and the displacement/pressure coefficient during coughing showed a significant increasing trend during pregnancy, but no changes were seen during the Valsalva manoeuvre. No relationship was found between subjective and objective incontinence data and the position and mobility of the urethro-vesical junction.

**Conclusion** The prevalence of incontinence in nulliparous women as found by the pad test was significantly higher in pregnancy (20%) than in the non-pregnant control group (4%). Perineal ultrasound of the urethro-vesical junction showed lowering of the pelvic floor occurring as early as 12-16 weeks of pregnancy. Serial measurements of the displacement/pressure coefficient suggest that the dynamic characteristics of the connective tissues of the pelvic floor remain unaltered, whereas a significant decrease in pelvic floor muscle contraction occurs. Since no relation was found between measurements of the urethro-vesical junction and incontinence, urinary incontinence in pregnancy is most likely explained by other factors.

## INTRODUCTION

It is generally accepted that as pregnancy proceeds a growing number of women complain of urinary incontinence. Accidental loss of urine is reported in 17-25% of women in early pregnancy<sup>1,2</sup> and in up to 36-67% in late pregnancy<sup>1,3</sup>. Moreover, the degree of incontinence worsens as pregnancy advances<sup>3</sup>. The prevalence of incontinence during pregnancy is usually calculated from self-reporting of symptoms without objective measurements. Urinary incontinence during pregnancy is ascribed to detrusor instability resulting from changes in hormone levels<sup>4</sup> and to anatomical changes due to the growing uterus and the engagement of the fetal head in the pelvis<sup>5</sup>. Others<sup>6</sup> have suggested that reduced fascial strength and subsequent weakness of the pelvic supports might contribute to urinary incontinence during pregnancy.

The scarcity of information on urinary incontinence during pregnancy, the wide variation of the reported prevalence and the uncertainty concerning the underlying mechanism prompted us to investigate these issues in a prospective longitudinal study. In a homogeneous cohort of nulliparous pregnant women, urinary incontinence was assessed by questionnaire, and by a 24-hour pad test. Changes in pelvic floor function during the course of pregnancy were dynamically investigated by perineal ultrasound measurements of the position and the mobility of the urethro-vesical junction.

## METHODS

One hundred and seventeen women who attended the outpatient clinics of the University Hospital Groningen and the Martini Hospital Groningen enrolled for the study. The women ranged in age from 17 to 41 years (mean and median 30 years). All women were of Caucasian origin, except three who were of Mediterranean origin. All women were nulliparous and had no history of incontinence, pelvic operations or neurological disease. Written informed consent was obtained from all participating women. The study was approved by the medical ethical committees of both hospitals. For this study the women were investigated three times, at 12-16 weeks, at 28-32 weeks and at 36-38 weeks of pregnancy. During the study six women withdrew because of matters of inconvenience, and three had their delivery

preceding the third visit. To investigate changes early in pregnancy an age-matched control group of 27 nulliparous non-pregnant continent women from the infertility outpatient clinic was included. These non-pregnant controls were subjected to the same protocol, including the pad test, but only once.

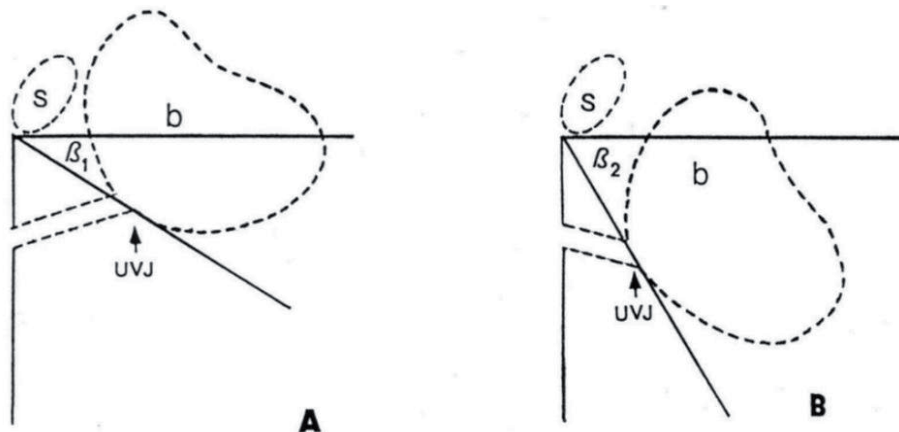
At each visit all women completed a questionnaire on symptoms of incontinence. Stress incontinence was defined as the loss of urine on physical effort and urge incontinence as the loss of urine associated with a strong desire to void. If incontinence was reported women tried to classify it as stress or urge incontinence. At the first visit pads were given to all women, to be worn for 24 hours preceding the second and third visit. Three pads were packed in a plastic bag and weighed by the investigators before and after use. The women received a written instruction and were free to wear one, two or three pads. It was emphasised that the bag should be closed carefully every time a pad was changed to prevent evaporation. If the bag was returned open or contained less than 3 pads the test was excluded from evaluation. The outcome of the 24-hour pad test was recorded as the weight gain at every visit of the pads. In accordance with the literature a cut-off level of 9 g was used to classify a woman as incontinent<sup>7,8</sup>.

Perineal ultrasound was performed with an Aloka 600 machine with a 3.5 MHz convex scanner and the woman in the lithotomy position. Bladder volume varied from 100-300 ml. The position of the urethro-vesical junction was recorded continuously during coughing and during the Valsalva manoeuvre. The central part of the curved array probe was placed at the same level as the inferior border of the symphysis, with the radial line kept horizontally. Care was taken to prevent rotation of the convex ultrasound probe in the sagittal plane and to place the probe only lightly against the perineum during the various procedures. Simultaneous abdominal pressure changes were recorded by the insertion of a microtip pressure transducer (Gaeltec) inserted high in the posterior fornix of the vagina<sup>9</sup>. At a frequency of 8Hz ultrasound images and pressure measurements were recorded on line and stored on a hard disk during each exercise after the pre-set trigger pressure level of the system had been met by the participant. During coughing this level was 70 cm H<sub>2</sub>O and during Valsalva 50 cm H<sub>2</sub>O. Off line, the displacement of the urethro-vesical junction was measured with respect to the fixed lower edge of the symphysis and was expressed in degrees, as shown in Figure 1. Like others we defined the position of the bladder neck as the angle  $\beta_{1,2}$  between the line going from the urethro-vesical junction to the inferior border of the pubic bone and a reference line<sup>10-12</sup>.

As a reference line we used the radial line from the curved array probe through the horizontal plane that goes through the inferior border of the pubic bone.

The effect of possible changes in the position of the ultrasound probe relative to the pelvis as a result from unintended rotational movements during coughing and during the Valsalva manoeuvre was investigated in ten women (nos. 10, 20...100). For this purpose, a line from the inferior border of the pubic bone through the center of the pubic bone was added. In this way in each participant a fixed line was obtained. The variation of the angle between this fixed line and our reference line is an index of rotational artefacts. The standard deviation for this angle during a session (24 samples), measured in 10 women, was 1.6 degrees on average for coughing and 2.4 degrees for the Valsalva manoeuvre. Comparison of the values of this angle at the consecutive measurements during the course of pregnancy revealed no significant differences. Also, the distance between the urethro-vesical junction and the inferior pubic bone showed no significant changes during a session, the average distance being 46.5 mm (SD 3.0 mm) during coughing and during the Valsalva manoeuvre.

**Figure1:** Schematic representation of measurement of the angle of the urethro-vesical junction.



*A at rest, B during Valsalva or coughing.*  
*UVJ = Urethro Vesical Junction, S = symphysis, b = bladder*

During the Valsalva manoeuvre and during coughing a set of time related data of displacement of the urethro-vesical junction and the change in abdominal pressure was obtained, as shown in Figure 2a and 2b. From these two simultaneously

recorded variables an X-Y graph was plotted, showing the relationship between displacement of the urethro-vesical junction and abdominal pressure (Figure 2c). The slope of the line connecting the starting point and the point at maximum pressure was considered as a characteristic index of pelvic floor function. We called this the displacement/pressure coefficient, which is expressed as displacement of the urethro-vesical junction in degrees per abdominal pressure change in cm H<sub>2</sub>O. Measurements of angles and pressures in each session were done in duplicate. Since the results were not available in real time, flaws were not detected until the data were analysed. To remove outliers from the data we excluded from further analysis those sessions with duplicates outside the 95% confidence interval<sup>13</sup>. The coefficient of variation within each session was < 4% for angles ( $A_0$  and  $A_{max}$ ) and the corresponding pressures ( $P_0$  and  $P_{max}$ ). For the displacement/pressure coefficient the variation coefficient was 14% in the Valsalva and 17% in the cough experiments.

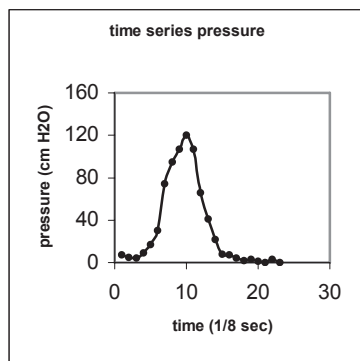


Figure 2a

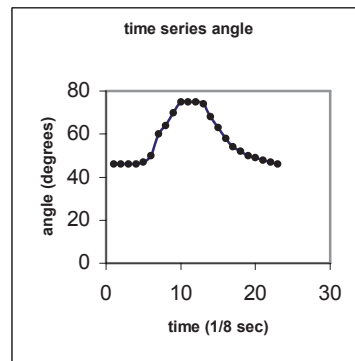


Figure 2b

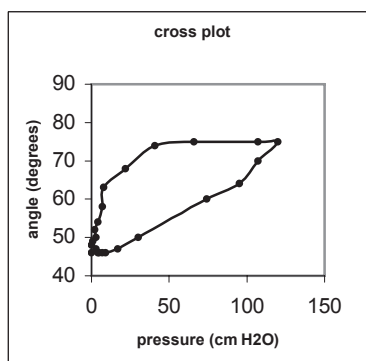


Figure 2c

**Figure 2a-2c:** Simultaneous perineal ultrasound and vaginal pressure measurements. Figure 2a shows the time series for pressure changes during the Valsalva manoeuvre, at a frequency of 8 Hz. Figure 2b shows the simultaneous recorded time series for the rotation angle and figure 2c is the cross plot of figure 2a and 2b.

$A_0$  is the resting angle, the rotation angle at rest, pressure is minimal.  $A_{max}$  is the maximal angle, the rotation angle at maximal pressure during Valsalva. The lower part of the curve reflects the angle with increasing vaginal pressure whereas the upper part of the curve reflects the angle with decreasing vaginal pressure. The slope of the line connecting the starting point and the point at maximum pressure is defined as a characteristic parameter of the pelvic floor, the displacement/pressure coefficient, expressed as the displacement of the urethro-vesical junction in degrees per abdominal pressure change in cm H<sub>2</sub>O.

## STATISTICAL ANALYSIS

The Kolmogorov-Smirnov test was used to assess if the data had a Gaussian distribution. The progressive increase in incontinence during pregnancy was verified with the chi-squared test for trend. For the weight gain in the pad test the Mann-Whitney test was used and for 2 \* 2 frequency tables Fisher's exact test. Friedman's two-way analysis of variance by ranks was used for the longitudinal ultrasound and pressure measurements, and the Mann-Whitney test for comparison between these measurements in non-pregnant and early pregnant women.

## RESULTS

### *Incontinence during pregnancy*

During pregnancy the number of women reporting urinary incontinence increased significantly from 16% at 12-16 weeks, to 30% at 28-30 weeks and to 35% at 36-38 weeks ( $p=0.002$ ). Of the women reporting urinary incontinence, stress incontinence occurred in 45, 49 and 59% respectively, at the first, the second and the third visit. The remaining women reported urge incontinence. Pads were returned according to the protocol by 98% and 86% of the women at the second and third visit respectively. The mean weight gain of the pads was 5.7 gram, at 28-32 weeks and at 36-38 weeks gestation. Using a definition of stress incontinence of weight gain of 9 grams, a significant association ( $p=0.03$ ) was found between self-reported incontinence and the results of the pad test. In the early pregnant group the pad test revealed significantly more weight gain than was found in the non pregnant control group (5.7g vs. 3.1g,  $p<0.001$ ). Comparison of the women with stress and urge incontinence showed no significant differences in results of the pad tests.

### *Measurements of the urethro-vesical junction in pregnancy*

The resting angle of the urethro-vesical junction ( $A_0$ ) widened significantly during pregnancy, from 51.5 degrees at 12-16 weeks to 62.0 degrees at term ( $p<0.001$ ). At 12-16 weeks the resting angle was already significantly increased as compared to that in the non-pregnant control group, where a resting angle of the urethro-vesical junction of 44.5 degrees was found ( $p=0.01$ ). The effects of coughing and of the Valsalva manoeuvre on the mobility of the urethro-vesical junction were investigated



by using the displacement /pressure coefficient. During coughing and during the Valsalva manoeuvre, the mean peak pressures that were reached were 88 and 75 cm H<sub>2</sub>O, respectively. During coughing there was a significant increasing trend in the displacement/pressure coefficient during of pregnancy ( $p=0.03$ ), although at 12-16 weeks the difference was not significant from the non-pregnant control group. For the Valsalva manoeuvre no significant change in the displacement/pressure coefficient was found at 12-16 weeks compared with the control group, 0.32 vs. 0.30, and throughout pregnancy no significant difference was observed (Tables 1 and 2).

No relationship was found between measurements of the urethro-vesical junction and the women's perception of urinary incontinence. This applied to all variables studied during coughing as well as during the Valsalva manoeuvre: the angle of the urethro-vesical junction at rest, the angle of the urethro-vesical junction at maximum pressure and the displacement/pressure coefficient. Also, no statistically significant relationship could be demonstrated between these variables and the results of the pad tests.

**Table 1:** Relationship between the angle of the urethro-vesical junction and pressure during coughing at consecutive visits in pregnancy, and in the controls

	Rest resting angle ( $A_0$ )		Cough maximal angle ( $A_{max}$ )		Pressure change ( $\Delta P$ )		displacement/ pressure coefficient ( $A_{max} - A_0$ ) / $\Delta P$	
	median	$\Sigma$ ranks*	median	$\Sigma$ ranks*	median	$\Sigma$ ranks <sup>§</sup>	median	$\Sigma$ ranks*
12-16 weeks	51.5	146.5	65.0	150.0	89.0	207.5	0.16	176.0
28-32 weeks	58.5	206.5	74.5	212.0	89.5	216.0	0.17	199.5
36-38 weeks	62.0	241.0	79.0	238.0	85.5	188.5	0.17	218.5
-	-	-	-	-	-	-	-	-
controls	44.5	-	62.5	-	94.0	-	0.16	-

Angles in degrees, pressure in cm H<sub>2</sub>O and displacement/pressure coefficient in degrees/ cm H<sub>2</sub>O, medians and sum of ranks, \* significant trend,  $p < 0.01$ , two way analysis of variance in ranks, <sup>§</sup> ns.

**Table 2:** Relationship between the angle of the urethro-vesical junction and pressure during the Valsalva manoeuvre at consecutive visits in pregnancy, and in controls.

	Rest resting angle ( $A_0$ )		Valsalva maximal angle ( $A_{max}$ )		Pressure change ( $\Delta P$ )		displacement/ pressure coefficient ( $A_{max} - A_0$ ) / $\Delta P$	
	median	$\Sigma$ ranks*	median	$\Sigma$ ranks*	median	$\Sigma$ ranks*	median	$\Sigma$ ranks <sup>§</sup>
12-16 weeks	49.5	122	76.6	135	81.6	184	0.32	157
28-32 weeks	57.1	173	83.6	179	76.8	179	0.32	170
36-38 weeks	61.5	203	84.9	184	8.8	147	32.32	171
-	-	-	-	-	-	-	-	-
controls	43.5	-	73.5	-	77.0	-	0.30	-

Angles in degrees, pressure changes in cm and displacement/pressure coefficient in degrees/ cm H<sub>2</sub>O, medians and sum of ranks, \* significant trend,  $p < 0.01$ , two way analysis of variance in ranks, <sup>§</sup> ns.

## DISCUSSION

The prevalence of incontinence reported by the pregnant women in our study is in agreement with data from the literature, and so is the increase observed during the course of pregnancy. However, the reliability of figures on the prevalence of incontinence in pregnancy, based on self-reporting of urinary incontinence, has been questioned. Burgio et al. found that the self-reported prevalence of incontinence in pregnancy was associated among other things with the level of education and attendance at childbirth classes<sup>14</sup>. Therefore they recommended self-reporting of symptoms should be supplemented by objective measures, such as the pad test. Ryhammer et al., in a study in menopausal women found no relation between self-reported incontinence and objective measures such as the 24-hour home pad test<sup>15</sup>.

If we use a cut-off point of 9 grams weight gain in the 24-hour pad test to define urinary incontinence<sup>7,8</sup>, a significant relation was found between the results of the pad test and the self-reporting of symptoms. Thus our findings indicate that there is a relation between objective and subjective measurements of incontinence in pregnancy.

By using the 9 gram cut-off of the 24-hour pad test to define urinary incontinence the frequency of incontinence in our study was 24% and 16% at 12-16 and 36-38 weeks respectively. These figures are still significantly higher than in the non-pregnant control group where only one out of 27 women (4%) had a pad test result of 9 grams or more. Pregnancy itself therefore seems to be the causal factor for incontinence, although the mechanism is not clear.

The function of the pelvic floor is thought to play an important part in the mechanism of continence in women, and so we investigated the changes in pelvic floor function during pregnancy and its relation to incontinence. We used simultaneous recording of abdominal pressure and the consequent changes in the position of the urethro-vesical junction. The method of perineal ultrasound to study vesical neck mobility has been evaluated by Schaer et al. who found it to be valid<sup>16</sup>. Like most other investigators, we have applied perineal ultrasound in the lithotomy position. From the study of Vierhout and Jansen<sup>17</sup> it is evident that there is a small but consistent change in the localisation of the bladder neck when moving from the lithotomy position to the vertical position. Although there is a difference in the starting-point of the localisation of the urethro-vesical junction, the direction and the magnitude

of the displacement during Valsalva and coughing is not essentially different whether the woman is standing or in the lithotomy position. In practice, it appears more difficult to obtain accurate measurements in the vertical position. Moreover, Vierhout and Jansen found that women experience more discomfort during such measurements in the standing position than in the lithotomy position.

The proximal urethra and the anterior vaginal wall are intimately connected and attached to the muscles of the pelvic diaphragm and to the arcus tendineus fasciae pelvis. Support of the urethra at rest comes from both its attachments to the arcus tendineus fasciae pelvis and the resting tone of the muscles of the pelvic diaphragm<sup>18</sup>. Therefore, the mobility of the urethro-vesical junction can be used as an index of the mobility of these pelvic floor structures. Already in early pregnancy we found a significant widening of the resting angle compared with the non-pregnant control group. This finding is interpreted as lowering of the pelvic floor, a phenomenon previously described in late pregnancy by Peschers et.al.<sup>5</sup>. They explain it by the pressure of the growing uterus and the engagement of the fetus and by the hormonal changes of pregnancy. As we observed the widening already early in pregnancy, it seems unlikely that pressure by the growing uterus and the engaging fetus are the only factors responsible for the observed widening of the angle of the urethro-vesical junction. We therefore believe that the effects of pregnancy hormones on the connective tissues of the pelvic floor are much more important in this respect. This is supported by a study of Landon et al<sup>6</sup>. They showed intrinsic tissue changes due to the hormonal state of pregnancy itself, resulting in an increased stretching of fascial tissue. Versi et al found that the collagen content of skin is associated with several characteristics of the urethral pressure profile, both at rest and during coughing. They state that urethral collagen might well be under similar hormonal and genetic control mechanisms as skin collagen<sup>19</sup>. The exact way in which pregnancy and its hormones affect the mechanical properties of the supporting connective tissue and fascia of the urogenital tract in the human is unknown. In dogs elevated levels of progesterone in pregnancy decrease urethral pressure by facilitating  $\beta$ -adrenergic responses<sup>20</sup>. In the human progesterone receptors have been demonstrated and quantified in the female pelvic floor muscles, the urogenital ligaments and in the myometrium by monoclonal antibody assay techniques<sup>21</sup>. It is likely that in pregnancy high levels of progesterone lead to hypotonicity of the pelvic floor structures.

The relationship between the position of the urethro-vesical junction and abdominal pressure, the displacement/pressure coefficient, provides quantitative insight into

the functional dynamic state of the supportive structures of the urethro-vesical junction, i.e. the pelvic floor muscles and adjacent connective tissue. We found that the displacement /pressure gradient remained virtually constant with the Valsalva manoeuvre during the course of pregnancy. This observation suggests that the dynamic connective tissue qualities of the pelvic floor are not progressively influenced by pregnancy. Unlike the Valsalva manoeuvre, coughing is accompanied by a reflex contraction of pelvic floor muscles, which makes the pelvic floor more resistant to deformation during coughing than during the Valsalva manoeuvre. This explains why with coughing the displacement/pressure coefficient is only half the value of this coefficient with the Valsalva manoeuvre. During the course of pregnancy the increase of the displacement/pressure coefficient during coughing indicates a decreasing effect of contraction of the pelvic floor muscles. This may be a direct consequence of impaired muscle contraction due to muscular or nerve fibre tissue changes. However, it is also possible that we measured an altered effect of normal muscle contraction, primarily resulting from connective tissue changes. A combination of these factors is possible. Much electrophysiological research has been done in incontinent women and in women with genital prolapse. In nerve conduction studies nerve damage, possibly as a result of delivery, with resultant pelvic floor muscle weakness is a common finding in women with stress incontinence of urine<sup>22</sup>. However, it seems unlikely that during pregnancy, prior to labour and delivery, that nerve damage and denervation plays an important role. Some investigators suggest that altered innervation does not play an important part in the aetiology of stress incontinence. The reason for this may be that minor alterations in the innervation of striated muscle are not related to a reduction in muscle power, and are therefore not of clinical or aetiological significance in incontinence<sup>23</sup>. Electromyography of pelvic floor muscles during pregnancy would be required to clarify this issue.

Recently, King et al, also using perineal ultrasound, reported that primigravid women with postpartum urinary stress incontinence had greater mobility of the bladder neck antenatally than women who were continent postpartum<sup>24</sup>. Although they found a high incidence of reported urinary incontinence antenatally, they do not mention a possible relationship between antenatal incontinence and mobility of the bladder neck antenatally. They suggest a constitutional susceptibility to stress incontinence brought about by a pre-existing collagen deficiency, exacerbated by increased collagen remodelling during pregnancy.

The results of the present study indicate that the changes in the quality of the

connective tissue or muscle of the pelvic floor are not responsible for the higher prevalence of urine incontinence in pregnancy. Our study focussed on the changes in the extrinsic mechanism of continence, pelvic floor muscle and fascia. Changes in the intrinsic mechanism of continence, the urethral tissues including the urethral sphincter, during pregnancy were studied by van Geelen<sup>25</sup> and Iosif<sup>26</sup>. They concluded that an inherent weakness of the urethral sphincter mechanism could largely explain the occurrence of incontinence in pregnancy. Their conclusion is indirectly supported by the results of this present study which revealed that changes in the extrinsic continence mechanism are not related to urinary incontinence in the course of pregnancy. We are currently investigating the possible relationship between the antenatal findings and the effects of labour and delivery on the mobility of the urethro-vesical junction postnatally and urinary incontinence.

## REFERENCE LIST

- (1) Stanton SL, Kerr-Wilson R, Harris VG. The incidence of urological symptoms in normal pregnancy. *Br J Obstet Gynaecol* 1980; 87(10):897-900.
- (2) Cardozo L, Cutner A. Lower urinary tract symptoms in pregnancy. *Br J Urol* 1997; 80 Suppl 1:14-23.
- (3) FRANCIS WJ. The onset of stress incontinence. *J Obstet Gynaecol Br Emp* 1960; 67:899-903.
- (4) Miodrag A, Castleden CM, Vallance TR. Sex hormones and the female urinary tract. *Drugs* 1988; 36(4):491-504.
- (5) Peschers U, Schaer G, Anthuber C, DeLancey JO, Schuessler B. Changes in vesical neck mobility following vaginal delivery. *Obstet Gynecol* 1996; 88(6):1001-1006.
- (6) Landon CR, Crofts CE, Smith ARB, Trowbridge EA. Mechanical properties of fascia during pregnancy: a possible factor in the development of stress incontinence of urine. *Contemp Rev Obstet Gynaecol* 1990; 2:40-46.
- (7) Versi E, Orrego G, Hardy E, Seddon G, Smith P, Anand D. Evaluation of the home pad test in the investigation of female urinary incontinence. *Br J Obstet Gynaecol* 1996; 103(2):162-167.
- (8) Lose G, Jorgensen L, Thunedborg P. 24-hour home pad weighing test versus 1-hour ward test in the assessment of mild stress incontinence. *Acta Obstet Gynecol Scand* 1989; 68(3):211-215.

- (9) James ED, Niblett PG, MacNaughton JA, Shaldon C. The vagina as an alternative to the rectum in measuring abdominal pressure during urodynamic investigations. *Br J Urol* 1987; 60(3):212-216.
- (10) Chen GD, Su TH, Lin LY. Applicability of perineal sonography in anatomical evaluation of bladder neck in women with and without genuine stress incontinence. *J Clin Ultrasound* 1997; 25(4):189-194.
- (11) Mouritsen L, Strandberg C. Vaginal ultrasonography versus colpo-cysto-urethrography in the evaluation of female urinary incontinence. *Acta Obstet Gynecol Scand* 1994; 73(4):338-342.
- (12) Meyer S, De Grandi P, Schreyer A, Caccia G. The assessment of bladder neck position and mobility in continent nullipara, multipara, forceps-delivered and incontinent women using perineal ultrasound: a future office procedure? *Int Urogynecol J Pelvic Floor Dysfunct* 1996; 7(3):138-146.
- (13) Bland JM, Altman DG. Statistical methods for assessing agreement between two methods of clinical measurement. *Lancet* 1986; 1(8476):307-310.
- (14) Burgio KL, Locher JL, Zyczynski H, Hardin JM, Singh K. Urinary incontinence during pregnancy in a racially mixed sample: characteristics and predisposing factors. *Int Urogynecol J Pelvic Floor Dysfunct* 1996; 7(2):69-73.
- (15) Ryhammer AM, Laurberg S, Djurhuus JC, Hermann AP. No relationship between subjective assessment of urinary incontinence and pad test weight gain in a random population sample of menopausal women. *J Urol* 1998; 159(3):800-803.
- (16) Schaer GN, Koechli OR, Schuessler B, Haller U. Perineal ultrasound for evaluating the bladder neck in urinary stress incontinence. *Obstet Gynecol* 1995; 85(2):220-224.
- (17) Vierhout ME, Jansen H. Supine and sitting rectal ultrasound of the bladder neck during relaxation, straining and squeezing. *Int Urogynecol J Pelvic Floor Dysfunct* 1991; 2:141-143.
- (18) DeLancey JO. Correlative study of paraurethral anatomy. *Obstet Gynecol* 1986; 68(1):91-97.
- (19) Versi E, Cardozo L, Brincat M, Cooper D, Montgomery J, Studd J. Correlation of urethral physiology and skin collagen in postmenopausal women. *Br J Obstet Gynaecol* 1988; 95(2):147-152.
- (20) Raz S, Zeigler M, Caine M. The effect of progesterone on the adrenergic receptors of the urethra. *Br J Urol* 1973; 45(2):131-135.
- (21) Smith P. Estrogens and the urogenital tract. Studies on steroid hormone receptors and a clinical study on a new estradiol-releasing vaginal ring. *Acta Obstet Gynecol Scand Suppl* 1993; 157:1-26.

- (22) Smith AR, Hosker GL, Warrell DW. The role of pudendal nerve damage in the aetiology of genuine stress incontinence in women. *Br J Obstet Gynaecol* 1989; 96(1):29-32.
- (23) Barnick CG, Cardozo LD. Denervation and re-innervation of the urethral sphincter in the aetiology of genuine stress incontinence: an electromyographic study. *Br J Obstet Gynaecol* 1993; 100(8):750-753.
- (24) King JK, Freeman RM. Is antenatal bladder neck mobility a risk factor for postpartum stress incontinence? *Br J Obstet Gynaecol* 1998; 105(12):1300-1307.
- (25) van Geelen JM, Lemmens WA, Eskes TK, Martin CB, Jr. The urethral pressure profile in pregnancy and after delivery in healthy nulliparous women. *Am J Obstet Gynecol* 1982; 144(6):636-649.
- (26) Iosif S, Ulmsten U. Comparative urodynamic studies of continent and stress incontinent women in pregnancy and in the puerperium. *Am J Obstet Gynecol* 1981; 140(6):645-650.



# CHAPTER 3

## Anatomical and functional changes in the lower urinary tract following spontaneous vaginal delivery

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## ABSTRACT

**Objective** To assess the incidence of urinary incontinence in pregnancy and after spontaneous vaginal delivery and its relation with changes in the static and dynamic function of the pelvic floor.

**Design** Prospective longitudinal study.

**Setting** University Hospital and Martini Hospital Groningen, the Netherlands.

**Population** A cohort of 62 women before and after spontaneous vaginal delivery at term and 27 nulliparous non-pregnant controls.

**Methods** Urinary incontinence was measured by a questionnaire and by a 24-hour pad test. The position and mobility of the urethro-vesical junction were measured by perineal ultrasound and related to simultaneously measured abdominal pressure changes. Serial investigations were done at 38 weeks gestational age and at 6 weeks and 6 months after delivery

**Main outcome measure** Urinary incontinence and its relation with the position of the urethro-vesical junction at rest and with the mobility of the urethro-vesical junction during Valsalva and during coughing, indicated by the displacement/pressure coefficient and with obstetric variables.

**Results** After delivery, reported urinary incontinence was reduced from 26% at 38 weeks of gestation to 16% and 15% at six weeks and six months after delivery, respectively. Even lower rates were measured by the 24-hour pad test which revealed a decrease from 14 % at 38 weeks to 10% and 5% at 6 weeks and 6 month postpartum, respectively. Six weeks and 6 months after delivery the angle of the urethro-vesical junction at rest was significantly increased compared to the non-pregnant control women. Compared to the antenatal measurements, the displacement/pressure coefficients during coughing and during the Valsalva manoeuvre were significantly increased six weeks after delivery. Six months after delivery, only the coefficient for coughing was still significantly greater than the antenatal value and the value in the non-pregnant control group. No relations were found between urethro-vesical junction measurements and obstetric variables and subjective or objective urinary incontinence parameters.

**Conclusion** Though pregnancy and spontaneous vaginal delivery significantly increased the degree of bladder neck descent and permanently affected the function of the pelvic floor during coughing, urinary incontinence, quite common during pregnancy, disappears post partum in most women.

## INTRODUCTION

Pregnancy and delivery cause transient urinary incontinence in a considerable number of women<sup>1-3</sup>. Until now, it is not clear to what extent pregnancy itself or vaginal delivery contributes to the development of urinary incontinence. Following vaginal delivery neuromuscular damage and bladder neck hypermobility, indicating a change in pelvic floor function, has been confirmed<sup>4,5</sup>. Nevertheless in the great majority of women the incontinence has disappeared six months after delivery<sup>6,7</sup>. This prompted us to investigate in a longitudinal study the effects of pregnancy and delivery and puerperium on pelvic floor function in nulliparous women. Using perineal ultrasound, the position and mobility of the urethro-vesical junction were serially investigated. In a previous study, we examined changes in the position and mobility of the urethro-vesical junction during pregnancy in relation to incontinence in a group of nulliparous women<sup>8</sup>.

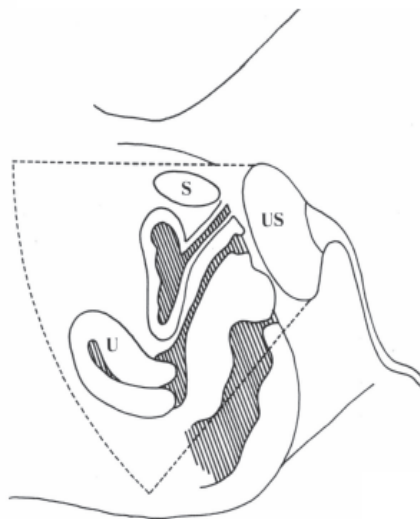
In the present study we investigated those women of the previous study who had a spontaneous vaginal delivery. We describe how spontaneous vaginal delivery affects the position and mobility of the urethro-vesical junction in relation to incontinence and whether these effects were still present after 6 months.

## METHODS

This study is part of a larger prospective study on the effect of pregnancy and delivery and puerperium on changes in pelvic floor mobility and urinary incontinence. Recently, we reported the effects of pregnancy<sup>8</sup>. Of the 117 nulliparous women, initially recruited for the study at the University Hospital Groningen and at the Martini Hospital Groningen, 62 had a spontaneous vaginal term delivery with vertex presentation. The median (range) duration of the first stage of labour was 430 (95-2340) minutes, while the median active stage of labour was 39 (6-147) minutes. A total of 16/62 (25%) women had an intact perineum; 22/62 (35%) had an episiotomy; 24/62 (39%) had perineal lacerations of several degrees, 22 1<sup>st</sup> and 2<sup>nd</sup> degree tears, two 3<sup>rd</sup> degree tears. The median (range) birth weight was 3487 (2100-4410) gram. The median (range) head circumference was 34 (31-37) centimetres. The effect of spontaneous vaginal delivery was investigated by serial analysis of data obtained antenatally at 36-38 weeks gestation, and at 6 weeks

postpartum. To study the effect of the puerperium measurements were taken 6 months postpartum. All women were nulliparous, with singleton pregnancies and none of them had a history of incontinence, pelvic operations or neurological disease before pregnancy. Data from 27 nulliparous non-pregnant women from the infertility outpatient clinic without a history of incontinence were included. These non-pregnant controls were subjected to the same study protocol, but only once. Written informed consent was obtained from all participating women. The medical ethical committees of both hospitals approved the study.

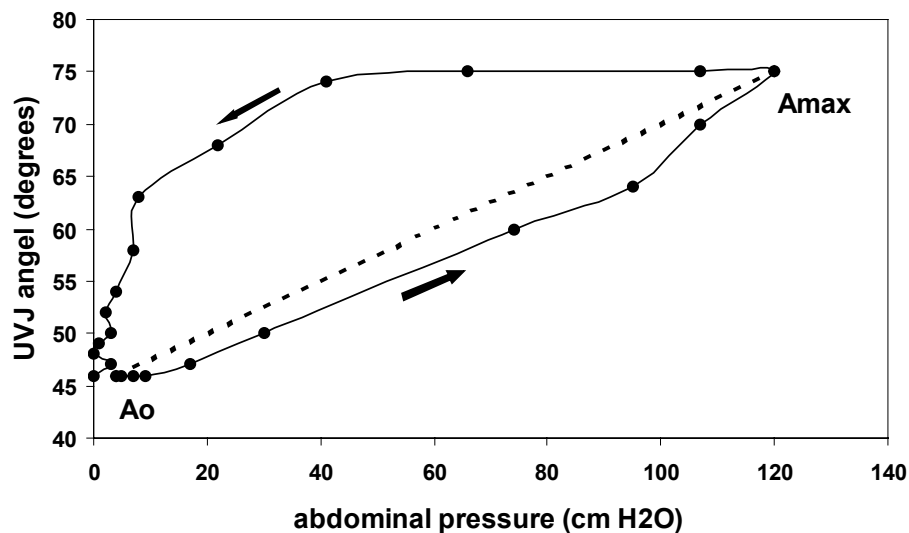
At each visit, all the women completed a questionnaire on symptoms of incontinence. They were also given pads to be worn for 24 hours preceding their visit. The outcome of the 24-hour pad test was recorded as the weight gain of the pad at each visit. A cut-off level of 9 g was used to classify a woman as incontinent.



**Figure 1:** Schematic representation of perineal ultrasound scanning of the UVJ with the woman in the lithotomy position. S = symphysis, U = uterus and US = ultrasound probe.

Perineal ultrasound was performed with Aloka 600 equipment with a 3.5 MHz convex transducer and the woman in the lithotomy position (Fig. 1). Bladder volume varied from 100-300 ml. The position of the urethro-vesical junction was recorded continuously during coughing and during the Valsalva manoeuvre. Simultaneous abdominal pressure changes were recorded by the insertion of a microtip pressure transducer (Gaeltec) high in the posterior fornix of the vagina. During the Valsalva manoeuvre and during coughing, sets of time-related data were obtained on the

displacement of the urethro-vesical junction and the change in abdominal pressure. A graph was plotted of these two simultaneously recorded variables to show the relation between displacement of the urethro-vesical junction and abdominal pressure changes (Fig. 2). The slope of the line connecting the starting point and the point of maximum pressure was considered to be a characteristic index of pelvic floor function. We called this the displacement/pressure coefficient; it is expressed as displacement of the urethro-vesical junction in degrees per abdominal pressure change in cm H<sub>2</sub>O. A more extensive description of the methods can be found in a previous article<sup>8</sup>.



**Figure 2:** Cross plot of intra-abdominal pressure and urethro-vesical junction (UVJ) angle measurement.  $A_0$  is the resting angle, the rotation angle at rest, pressure is minimal.  $A_{max}$  is the maximal angle, the rotation angle at maximal pressure during Valsalva, or coughing. The lower part of the curve reflects the UVJ angle at increasing pressure whereas the upper part of the curve reflects the angle during decreasing pressure. The slope of the line connecting the starting point and the point at maximum pressure is defined as the displacement/pressure coefficient, a characteristic index of the pelvic floor, expressed as the displacement of the urethro-vesical junction in degrees per abdominal pressure change in cm H<sub>2</sub>O.

## STATISTICAL ANALYSIS

The Kolmogorov-Smirnov test was used to assess if the data had a Gaussian distribution. For the weight gain in the pad test and for the UVJ measurements, the Mann-Whitney U test was used and for 2 \* 2 frequency tables Fisher's exact test.

To identify significant relationships between variables the Spearman correlation test was used.

**Table 1:** Serial measurements of UVJ-mobility, pad-test and reported incontinence.

	38 weeks pregnancy	6 weeks postpartum	6 months postpartum	Nulliparous Controls
Resting angle UVJ <sub>AO</sub>	67	66	57**	44.5***
Coefficient <sub>cough</sub>	0.20	0.25*	0.24**	0.16***
Coefficient <sub>Valsalva</sub>	0.30	0.39*	0.35	0.30
Positive pad-test	7/51	4/41	2/40	1/27
Reported incontinence	16/62	10/62	9/62	-

*Resting angle in degrees; Coefficient, displacement/pressure coefficient in degrees per cm H<sub>2</sub>O for cough and for Valsalva; positive pad-test, i.e. pad-test more than 9 grams. \* significant difference 6 wks pp compared to 38 wks, \*\*significant difference 6 months pp compared to 38 wks, \*\*\* significant difference controls compared to 6 months pp. Level of significance,  $p < 0.05$ .*

## RESULTS

### Urinary incontinence (Table 1)

Postpartum, the number of women reporting incontinence was reduced from 16/62 (26%) at 38 weeks of pregnancy to 10/62 (16%) at 6 weeks postpartum and to 9/62 (15 %) at 6 months postpartum.

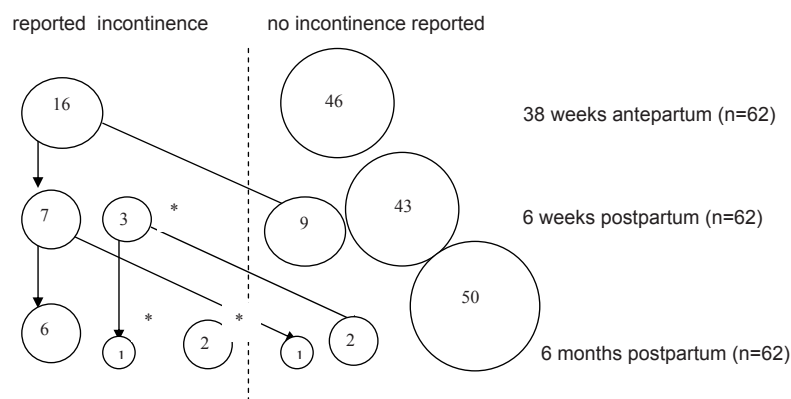
A positive pad test result, i.e. a weight gain of nine grams or more, was found in 7/51 (14%), 4/41 (10%) and 2/40 (5%) at the three consecutive measurements. De novo post partum incontinence was reported by 3 of the 9 women at 6 months after delivery. All the women with a positive pad test at 38 weeks gestation became negative 6 month post partum, whereas only two women with a negative test ante partum revealed a positive test at 6 months post partum. All the women who had a positive pad test at 38 weeks of pregnancy were also tested at 6 weeks and 6 months postpartum, whereas only 34 out of 44 of the women with a negative pad test antenatally turned in their pads post partum. Therefore the number of positive pad tests may be over represented. Moreover 33/35 women with a positive questionnaire (cumulative for three measurements) turned in their pads according to the protocol, whereas 103/151 women with a negative questionnaire did. Figures 3 and 4 show the sequential results of the subjectively reported incontinence and the pad tests from 38 weeks gestation to 6 months post partum.

**Measurements of the urethro-vesical junction (Table 1)**

The resting angle of the urethro-vesical junction ( $A_0$ ) remained hardly unchanged 6 weeks after vaginal delivery, but decreased significantly at six months postpartum, from 67 to 57 degrees, respectively. This value is significantly greater than the 44.5 degrees in the non-pregnant control group.

The effects of coughing and of the Valsalva manoeuvre on the mobility of the urethro-vesical junction were investigated by calculating the displacement/pressure coefficient. During coughing, the displacement/pressure coefficient increased significantly from 0.20 at 38 weeks of pregnancy to 0.25 at 6 weeks postpartum. At 6 months, the median coefficient was 0.24, which is still significantly higher than the antepartum value at 38 weeks. During the Valsalva manoeuvre, the median coefficient increased significantly from 0.30 at 38 weeks gestation to 0.39 at 6 weeks postpartum. At six months, the median coefficient was 0.35, which was not significantly different from the antepartum value at 38 weeks and also not different from that of the non-pregnant control group of 0.30.

None of the UVJ measurements were correlated to subjective or objective incontinence data.

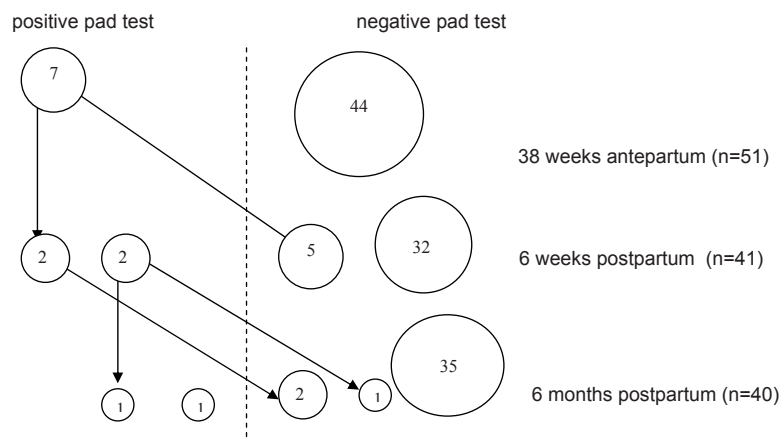


**Figure 3:** Longitudinal course of urinary incontinence as reported by questionnaire. The figures in the circles represent the number of women. 'De novo' incontinence is marked by an asterix

**Obstetric variables**

None of the obstetric variables, i.e. duration of the first stage of labour, duration of the active stage of labour, intact perineum, episiotomy, lacerations of several

degrees, birth weight and head circumference, were correlated with the incontinence outcome measurement or with measurements of the UVJ, or with changes in the individual outcome of these measurements.



**Figure 4:** Longitudinal course of incontinence diagnosed by the pad test. The figures in the circles represent the number of women.

## DISCUSSION

The results of the present study show a marked reduction in the incidence of urinary incontinence six weeks after delivery, as compared to 38 weeks of gestation. This holds for the reported as well as for the objectively measured incontinence by the pad test.

De novo post partum incontinence was reported by 3 of the 9 women affected at 6 months after delivery. All the women with a positive pad test at 38 weeks gestation became negative 6 months post partum, whereas only two women with a negative ante partum test revealed a positive test at 6 months post partum. Apparently incontinence during pregnancy is a different entity than urinary incontinence during the postpartum state.

The mostly temporary incontinence in pregnancy can be interpreted as a result of interaction between predisposing hereditary factors and uterine pressure upon the bladder, in combination with hormonal effect upon the suspension ligaments of the urethra<sup>9</sup>. Persisting incontinence post partum is mainly the result of changes

of the pelvic floor function and anatomy, due to delivery. The (partial) irreversibility of these changes may indicate why stress incontinence appearing for the first time after vaginal delivery has a more serious prognosis than incontinence developing during pregnancy<sup>6</sup>.

The present study demonstrates that spontaneous vaginal delivery causes transient as well as long lasting changes in the lower urinary tract. It seems likely that the long lasting changes that we observed six months after delivery will be permanent. Parturition alters urethral support and as a consequence, the position of the bladder neck at rest becomes permanently descended. These findings are in agreement with the results of Peschers et al<sup>10</sup> who found that the bladder neck was significantly lower at rest in women after vaginal delivery than in nulligravid controls. The changes in pelvic floor reaction to coughing, as measured by an increase of the displacement/pressure coefficient, indicates reduced pelvic floor stiffness due to coughing. This finding adds to the effect of pregnancy, where we observed already a significant decrease in pelvic floor stiffness during coughing<sup>8</sup>. These findings are most likely the consequence of impaired muscle contraction due to muscular or nerve fibre tissue changes.<sup>11;12</sup>. However, it is also possible that we measured an altered effect of normal muscle contraction primarily resulting from connective tissue defects<sup>13</sup>.

Peschers et al<sup>10</sup> found an increased reaction to the Valsalva manoeuvre six to ten weeks after vaginal delivery and conclude therefore that vaginal delivery alters vesical neck mobility. However we found that parturition has only a transient influence on the pelvic floor reaction to the Valsalva manoeuvre. This transient increase in the displacement/pressure coefficient means a decrease in resistance to deformation, e.g. decreased stiffness of the pelvic floor. As the pelvic floor can be considered as a composition of collagen tissue and neuromuscular tissue<sup>14</sup>, transient changes in the collagen tissue conditions are most likely to be responsible for this phenomenon, because during Valsalva, neuromuscular activity from the pelvic floor is negligible. Landon<sup>15</sup> described this phenomenon as stress relaxation, molecular reorganisation and fibre movement leading to the necessary adaptation of collagen tissue to permit physiological stretching during pregnancy and parturition.

Our findings confirm the results of Howard et al<sup>16</sup>, who found that the cough stiffness of the pelvic floor in nulliparous women was significantly greater than that in the primiparous continent women, whereas stiffness during Valsalva did not differ between these groups

King et al<sup>17</sup>, also using perineal ultrasound, found that antenatal bladder neck



mobility was greater in primigravid women with postpartum incontinence than in women who were continent postpartum. They suggest that collagen susceptibility to changes during pregnancy, measured by changes in bladder neck mobility, may predict post partum incontinence. Keane<sup>18</sup> proves that collagen tissue defects in nulliparous women are indeed related to genuine stress incontinence. Therefore we focussed especially on the relation between antenatal bladder neck mobility and post partum stress incontinence. Although we did find increased bladder neck mobility in early pregnancy<sup>8</sup> and near term (this study), we could not confirm the results of King. Neither at the various stages of pregnancy, nor at 6 weeks and 6 months post partum. Neither for objective nor for subjective incontinence. This might be due to the small number of women with post partum stress incontinence in our study. Moreover we used the displacement pressure co-efficient, they measured UVJ displacement at a standardised Valsalva manoeuvre for all patients, i.e. a pressure of 40 cm H<sub>2</sub>O.

We did not find any relation between incontinence measurements, urethro-vesical junction measurements and obstetric variables. This concurs with evidence found in other large clinical studies that obstetric factors do not form independent risk factors for postpartum incontinence<sup>19-21</sup>. In a recently published paper on urodynamics in pregnancy and after delivery, obstetric variables did not have any consistent effects on objective bladder function<sup>22</sup>.

Delivery, especially vaginal delivery, is thought to play a major role in the aetiology of urinary stress incontinence. Parturition is thought to cause damage to the structure and function of the pelvic organs<sup>1;23;24</sup>. However, incontinence generally disappears within 3 months postpartum<sup>1;3;6;7</sup>. Later in life, particularly after repeated delivery and after menopause, incontinence may recur<sup>3;24;25</sup>. Owing to this time lag between delivery and the late onset of urinary incontinence, it is almost impossible to prove a causal relation. In addition, the relation is troubled by lifetime experiences that can influence a woman's chance of developing urinary incontinence.

## CONCLUSION

It is concluded that pregnancy and spontaneous vaginal delivery significantly permanently alter the static condition of bladder neck descent. The dynamics of the pelvic floor are affected as well. This effect is temporary for Valsalva and

permanent for coughing. Urinary incontinence, quite common during pregnancy, disappears post partum in most women. No evidence was found for a significant association between pelvic floor function measurement and urinary incontinence, nor at 6 weeks, nor at 6 months postpartum. As the onset of symptoms of urinary incontinence later in life is thought to be due to the combined effect of occult trauma during pregnancy and delivery and the progression of neuropathy during lifetime<sup>4;24</sup> the persistent anatomical and functional changes in the lower urinary tract that we found, could play an important role in the aetiology of urinary stress incontinence later in life.

## REFERENCE LIST

- (1) Stanton SL, Kerr-Wilson R, Harris VG. The incidence of urological symptoms in normal pregnancy. *Br J Obstet Gynaecol* 1980; 87(10):897-900.
- (2) Cardozo L, Cutner A. Lower urinary tract symptoms in pregnancy. *Br J Urol* 1997; 80 Suppl 1:14-23.
- (3) FRANCIS WJ. The onset of stress incontinence. *J Obstet Gynaecol Br Emp* 1960; 67:899-903.
- (4) Sultan AH, Kamm MA, Hudson CN. Pudendal nerve damage during labour: prospective study before and after childbirth. *Br J Obstet Gynaecol* 1994; 101(1):22-28.
- (5) Meyer S, Schreyer A, De Grandi P, Hohlfield P. The effects of birth on urinary continence mechanisms and other pelvic-floor characteristics. *Obstet Gynecol* 1998; 92(4 Pt 1):613-618.
- (6) Viktrup L, Lose G, Rolff M, Barfoed K. The symptom of stress incontinence caused by pregnancy or delivery in primiparas. *Obstet Gynecol* 1992; 79(6):945-949.
- (7) Thorp JM, Jr., Norton PA, Wall LL, Kuller JA, Eucker B, Wells E. Urinary incontinence in pregnancy and the puerperium: a prospective study. *Am J Obstet Gynecol* 1999; 181(2):266-273.
- (8) Wijma J, Weis Potters AE, de Wolf BT, Tinga DJ, Aarnoudse JG. Anatomical and functional changes in the lower urinary tract during pregnancy. *BJOG* 2001; 108(7):726-732.
- (9) Iosif CS, Ingemarsson I. Prevalence of stress incontinence among women delivered by elective cesarian section. *Int J Gynaecol Obstet* 1982; 20(2):87-89.
- (10) Peschers U, Schaer G, Anthuber C, DeLancey JO, Schuessler B. Changes in vesical neck mobility following vaginal delivery. *Obstet Gynecol* 1996; 88(6):1001-1006.

- (11) Snooks SJ, Badenoch DF, Tiptaft RC, Swash M. Perineal nerve damage in genuine stress urinary incontinence. An electrophysiological study. *Br J Urol* 1985; 57(4):422-426.
- (12) Smith AR, Hosker GL, Warrell DW. The role of pudendal nerve damage in the aetiology of genuine stress incontinence in women. *Br J Obstet Gynaecol* 1989; 96(1):29-32.
- (13) Richardson AC, Edmonds PB, Williams NL. Treatment of stress urinary incontinence due to paravaginal fascial defect. *Obstet Gynecol* 1981; 57(3):357-362.
- (14) DeLancey JO. Correlative study of paraurethral anatomy. *Obstet Gynecol* 1986; 68(1):91-97.
- (15) Landon CR, Crofts CE, Smith ARB, Trowbridge EA. Mechanical properties of fascia during pregnancy: a possible factor in the development of stress incontinence of urine. *Contemp Rev Obstet Gynaecol* 1990; 2:40-46.
- (16) Howard D, Miller JM, DeLancey JO, Ashton-Miller JA. Differential effects of cough, valsalva, and continence status on vesical neck movement. *Obstet Gynecol* 2000; 95(4):535-540.
- (17) King JK, Freeman RM. Is antenatal bladder neck mobility a risk factor for postpartum stress incontinence? *Br J Obstet Gynaecol* 1998; 105(12):1300-1307.
- (18) Keane DP, Sims TJ, Abrams P, Bailey AJ. Analysis of collagen status in premenopausal nulliparous women with genuine stress incontinence. *Br J Obstet Gynaecol* 1997; 104(9):994-998.
- (19) Farrell SA, Allen VM, Baskett TF. Parturition and urinary incontinence in primiparas. *Obstet Gynecol* 2001; 97(3):350-356.
- (20) Wilson PD, Herbison RM, Herbison GP. Obstetric practice and the prevalence of urinary incontinence three months after delivery. *Br J Obstet Gynaecol* 1996; 103(2):154-161.
- (21) Viktrup L, Lose G. Epidural anesthesia during labor and stress incontinence after delivery. *Obstet Gynecol* 1993; 82(6):984-986.
- (22) Chaliha C, Bland JM, Monga A, Stanton SL, Sultan AH. Pregnancy and delivery: a urodynamic viewpoint. *BJOG* 2000; 107(11):1354-1359.
- (23) Handa VL, V. Protecting the pelvic floor: obstetric management to prevent incontinence and pelvic organ prolapse. *Obstetrics and gynecology* 1996; 88(3):470-478.
- (24) Snooks SJ, Swash M, Mathers SE, Henry MM. Effect of vaginal delivery on the pelvic floor: a 5-year follow-up. *Br J Surg* 1990; 77(12):1358-1360.
- (25) MacLennan AH, Taylor AW, Wilson DH, Wilson D. The prevalence of pelvic floor disorders and their relationship to gender, age, parity and mode of delivery. *BJOG* 2000; 107(12):1460-1470.



# CHAPTER 4

**Pelvic floor characteristics after spontaneous and operative vaginal delivery. Serial studies prior to labor up to six months postpartum.**

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## ABSTRACT

**Background** Increasing parity and operative vaginal delivery have been suggested to be responsible for pelvic floor trauma with subsequent appearance of long term urinary stress incontinence [1-5]. However, there is no general agreement regarding this concept, as these findings could not be confirmed in all studies[6-9]. Until now, the results of epidemiological studies are inconclusive, as no single event, such as mode of delivery, has been found responsible for the development of urinary incontinence [10-13]. From earlier studies [14,15] we concluded that pregnancy and spontaneous vaginal delivery significantly alter the static and dynamic condition of bladder neck mobility. The objective of this study is to compare spontaneous and operative vaginal delivery for pelvic floor characteristics and urinary incontinence.

**Methods** A cohort of 92 nulliparous women, followed throughout pregnancy up to 6 months postpartum, where 62 had a spontaneous vaginal delivery and 30 had an operative vaginal delivery. Bladder neck position and mobility were measured by perineal ultrasound; urinary incontinence was assessed by a questionnaire and by a 24-hour pad test. Women were studied at 38 weeks pregnancy, and 6 weeks and 6 months after childbirth, respectively.

**Results** Ante- and postpartum pelvic floor characteristics did not differ for spontaneous and operative vaginal delivery. Birth related factors predicting changes in pelvic floor function were not identified. Post partum (hyper)mobility of the UVJ is significantly correlated with ante partum (hyper)mobility.

**Conclusions** Extrinsic factors predicting pelvic floor dysfunction, such as birth related factors, were not identified. As we found that post partum compliance is significantly correlated with ante partum values, intrinsic factors, such as collagen characteristics, may be more important.

**Keywords** Pelvic floor, perineal ultrasound, urethral mobility, incontinence, vaginal delivery

## METHODS

This study is part of a larger prospective study on the effect of pregnancy, delivery and puerperium on changes in pelvic floor mobility and urinary incontinence. The effects of pregnancy and of spontaneous vaginal delivery have been previously

described [14,15]. Of the 117 nulliparous women, initially recruited for that study at the University Medical Centre Groningen and at the Martini Hospital Groningen, 62 had a spontaneous vaginal term delivery with vertex presentation, 30 had operative vaginal delivery with vertex presentation, 22 had vacuum delivery and 8 had forceps delivery. All these women completed follow-up until 6 months post partum.

After ruling out significant difference in variance within and between groups, vacuum and forceps delivery, these groups were taken together and redefined as the operative vaginal delivery group. 15 women had a Cesarean delivery. As the number of women with Cesarean delivery was small and the indications were heterogeneous (primary and secondary, arrest of labor in several states) we decided to exclude the results from this publication.

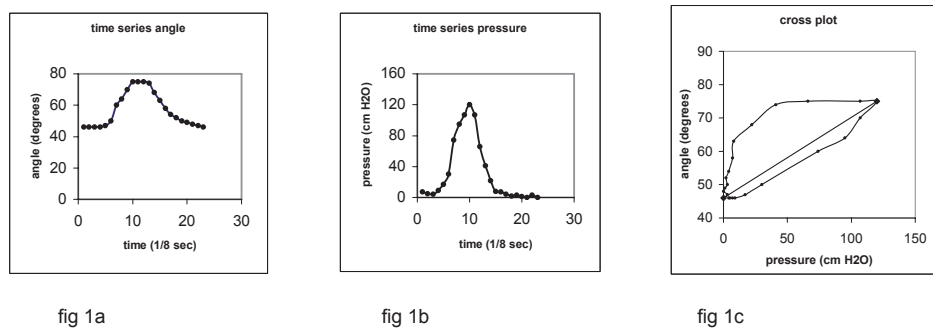
All women were nulliparous, with singleton pregnancies and none of them had a history of incontinence, pelvic operations or neurological disease before pregnancy. Written informed consent was obtained from all participating women. The study was approved by the medical ethics committee of the hospital.

Perineal ultrasound was performed with Aloka 600 equipment with a 3.5 MHz convex transducer and the woman in the lithotomy position. Bladder volume varied from 100-300 ml. The position of the urethro-vesical junction was recorded continuously during coughing and during the Valsalva maneuver. Simultaneous abdominal pressure changes were recorded by the insertion of a microtip pressure transducer (Gaeltec) high in the posterior fornix of the vagina. From the pressure changes and the simultaneously recorded bladder neck movements the displacement pressure coefficient was calculated, an index for the dynamic characteristics of the pelvic floor, known as compliance and complimentary to stiffness, see figure 1a-c. We refer to the displacement pressure coefficient as Valsalva and coughing compliance. All scans were made by the first and second author. Inter and intra observer variability was less than 5%. A more extensive description of the methods can be found in a previous article[15]. To remove outliers from the data we excluded from further analysis those sessions with duplicates outside the 95% confidence interval [16].

At each visit all women completed a questionnaire on symptoms of incontinence. Reported incontinence concerns the loss of urine on physical effort. They were also given pads to be worn for 24 hours preceding their second and following visits. The outcome of the 24-hour pad test was recorded as the weight gain of the pad at each visit. A cut-off level of 9 g was used to classify a woman as incontinent[17,18]

Using SPSS for windows, all continuous variables were tested for normal distribution

using the Kolmogorov-Smirnov test; no continuous variables were assigned to differ from the normal distribution. For comparison between groups of continuous variables the t-test for equality of means and for data in categories the Fisher's exact test was used. To identify significant relationships between variables the Pearson correlation test was used. ANOVA test was used to analyze variance between and within groups. Continuous data are presented as mean and standard error of the mean, categorical data are presented as absolute numbers and percentages.



**Figure 1a-c:** Simultaneous perineal ultrasound and vaginal pressure measurements. Figure 1a shows the time series for pressure changes during the Valsalva manoeuvre, at a frequency of 8 Hz. Figure 1b shows the simultaneous recorded time series for the rotation angle and figure 1c is the cross plot of figure 1a and 1b. The slope of the line connecting the starting point and the point at maximum pressure is defined as a characteristic parameter of the pelvic floor, the displacement/pressure coefficient, i.e. compliance, expressed as the displacement of the urethro-vesical junction in degrees per abdominal pressure change in cm H<sub>2</sub>O.

## RESULTS

*Birth related characteristics* for the study groups are presented in table 1. As may be expected, the number of episiotomies is higher in the operative vaginal delivery group. Other birth related parameters did not differ for the groups.

The results of the consecutive *measurements of the urethro-vesical junction* in the two study groups are presented in tables 2, 3 and 4.

### *The resting angle of the UVJ*

In the operative vaginal delivery group the resting angle of the urethro-vesical junction tended to be smaller than in the spontaneous group, but the difference did not reach the level of significance.

**Table 1:** Clinical characteristics of the study groups.

	Spontaneous delivery (n=62)	Operative vaginal delivery (n=30)	p-value
First stage of labor (min.)	555 (52)	555 (86)	0.975
Active stage of labor (min.)	46 (4)	60 (7)	0.065
Birth weight (grams)	3416 (62)	3440 (91)	0.810
Head circumference (cm.)	34.4 (0.2)	34.4 (0.3)	0.984
episiotomy	22	27	0.001
age (years)	30.3 (0.65)	31.0 (0.68)	0.477

**Table 2:** Resting angle (degrees) of the urethro vesical junction.

	Spontaneous vaginal delivery (n=62)	Operative vaginal delivery (n=30)	p-value	Mean difference (95% confidence limits)
38 wks	63 (1.9)	60 (2.8)	0.324	3.4 (-3.4 – 10.1)
6 wks	64 (1.7)	59 (2.9)	0.121	5.0 (-1.3 – 11.3)
6 mths	57 (1.9)	54 (3.6)	0.488	2.6 (-4.7 – 9.8)

**Table 3:** Compliance during coughing

	Spontaneous vaginal delivery (n=62)	Operative vaginal delivery (n=30)	p-value	Mean difference (95% confidence limits)
38 wks	0.20, (0.01)	0.17, (0.01)	0.129	0.028 (-0.008 – 0.065)
6 wks	0.26, (0.01)	0.25,(0.01)	0.730	0.008 (-0.038 – 0.054)
6 mths	0.25, (0.02)	0.21,(0.01)	0.136	0.037 (-0.012 – 0.087)

**Table 4:** Compliance during the Valsalva maneuver.

	Spontaneous vaginal delivery (n=62)	Operative vaginal delivery (n=30)	p-value	Mean difference, 95% confidence limits
38 wks	0.35, (0.02)	0.35, (0.04)	0.976	0.001 (-0.08 – 0.084)
6 wks	0.39, (0.02)	0.36,(0.03)	0.384	0.033 (-0.03 – 0.110)
6 mths	0.37, (0.03)	0.29,(0.03)	0.077	0.079 (-0.01 – 0.167)

***The compliance during coughing and during Valsalva***

Between the two groups no significant differences were found at the three points of time studied.

Both post partum Valsalva and cough compliance measurements at 6 weeks and 6



months were significantly correlated with the ante partum value as measured at 38 weeks of pregnancy, see table 5.

The results of the consecutive measurements in the two study groups for reported urinary incontinence and pad tests are given in tables 6 and 7.

*Urinary incontinence* was more frequently reported by women in the operative delivery group at all time points studied, but the results were not significantly different compared to the spontaneous delivery group. Pads were returned according to the protocol by 84% at term and 67% and 64% of the women at the first and second visit postpartum respectively. The results of the pad test showed a much lower incontinence rate, with similar values for the two groups.

The *obstetric variables* birth weight, head circumference, duration of stages of labor and condition of the post partum perineum did not significantly correlate with the pelvic floor characteristics that were measured. Also, no significant relationship was found between these obstetric variables and the reported urinary incontinence and the pad test results.

**Table 5:** Pearson correlation coefficients for cough and Valsalva compliances before and after delivery

	Compliance 6 wks post partum	Compliance 6 months post partum
Cough compliance ante partum	0.36 (p=0.01)	0.49 (p=0.001)
Valsalva compliance ante partum	0.39 (p=0.001)	0.45 (p=0.001)

**Table 6:** Number of women with self-reported incontinence, cross tabulation and relative risk

	Spontaneous delivery, (n=62)	Operative vaginal delivery, (n=30)	p-value	RR(95% confidence limits)
38 weeks	16 (26%)	12 (40%)	0.227	0.65(0.35-1.19)
6 weeks post partum	10 (16%)	9 (30%)	0.169	0.54(0.24-1.18)
6 months post partum	7 (11%)	7 (23%)	0.214	0.48(0.19-1.26)

**Table 7:** Number of women with a positive pad-test result and total number of returned pads according to the protocol, cross tabulation and relative risk

	Spontaneous delivery, (n=62)	Operative vaginal delivery, (n=30)	p-value	RR(95% confidence limits)
38 weeks	8/54(15%)	4/24(17%)	1.0	0.89(0.30-2.67)
6 weeks post partum	4/40(10%)	2/21(10%)	1.0	1.05(0.21-5.27)
6 months post partum	2/39(5%)	1/20(5%)	1.0	1.03(0.10-10.6)

## DISCUSSION

### *Pelvic floor characteristics*

In our study post partum (hyper) mobility is not influenced by the type of delivery, but by the ante partum mobility and therefore seems to be determined by (intrinsic) patient characteristics. As early as 1982 van Geelen found no relation between birth related factors and urethral pressure profile parameters, therefore he concluded at that time that an inherent weakness of the urethral sphincter mechanism plays a key role in the pathogenesis of stress incontinence[19]. Dietz and Steensma[20] found that women with a greater ante partum descent had most marked post partum change. King and Freeman[21] described a positive correlation between antenatal bladder neck mobility and post partum urinary incontinence possibly indicating that genetically defined collagen quality might play an important role. From a recent study Dietz[22] concludes that a significant genetic contribution to the phenotype of bladder neck mobility appears likely.

There is general belief that bladder neck descent is a predictor of genuine stress incontinence[23-25]. This means that persistent anatomical and functional changes in the pelvic floor may play an important role in the etiology of urinary stress incontinence later in life. Ultra fast MR Imaging of the pelvic floor anatomy has shown that pelvic floor laxity and supporting fascia abnormalities are most common in women with stress incontinence[26]. The importance of bladder neck mobility for the development of stress incontinence is emphasized by several authors[20,21,27].

In our study women with operative and spontaneous vaginal delivery have comparable ante partum and postpartum pelvic floor characteristics. In a previous study we reported significant changes due to childbirth for the spontaneous vaginal delivery group, i.e. a widening of the resting angle and increase of the compliance. We now found that operative vaginal delivery has comparable consequences. Therefore, if these changes are predictors of incontinence later in lifetime, in both groups women are at the same risk to develop incontinence later.

### *Obstetric variables*

Episiotomy is a routine procedure in operative vaginal delivery in our hospitals. We found no difference in pelvic floor characteristics after mediolateral episiotomy when compared to intact perineum or perineal lacerations. This matter is subject of debate for a long time already[28-30]. As the procedure and the indication for

episiotomy is not uniform in different countries, even not in different hospitals, it will be hard to find out the prognostic value for pelvic floor disorders. In our study it is not a prognostic factor for pelvic floor function later post partum. Other birth parameters, such as duration of labor, birth weight and head circumference did not correlate with pelvic floor characteristics as well. The finding that these extrinsic factors do not influence pelvic floor characteristics is in agreement with the conclusions of others who emphasize the influence of intrinsic factors, such as genetically determined characteristics of the collagen tissue[21,22,31].

#### *Incontinence parameters*

The present data showed no significant differences for urinary incontinence between the group of women who delivered spontaneously and the group who had an operative vaginal delivery. In a meta analysis the symptom of stress incontinence was 91% sensitive but only 51% specific for detecting genuine stress urinary incontinence as defined by the International Continence Society, based on history and urodynamic testing[32]. By using the 24 hour pad test we tried to bring forward objective data. We found a great difference between the number of women with self reported incontinence and incontinence according to a positive pad test. Urinary incontinence is measured twice as much by questionnaire as by pad test. The difference in outcome between the two methods is a well known phenomenon in the literature[33,34].

We realize that our study has its limitations. We did not investigate the effect of CS, so our conclusions are limited to vaginal birth. The weakness of the study is the relatively small number of patients; the strength is the prospective design, the homogeneous group, the objective measurements and the follow up until 6 months.

In conclusion we found that pelvic floor characteristics change as a result of vaginal delivery in nulliparous women. These changes occur after spontaneous as well as after operative vaginal delivery. As to these changes we found no significant differences between the spontaneously and the operatively delivered group. Also for urinary incontinence, both measured subjectively and objectively, we found no significant differences between the two groups of women. Extrinsic factors predicting pelvic floor dysfunction, such as birth related factors, were not identified. As we found that post partum compliance is significantly correlated with ante partum values, intrinsic factors, such as collagen characteristics, may be more important.

## REFERENCE LIST

- (1) Wilson PD, Herbison RM, Herbison GP. Obstetric practice and the prevalence of urinary incontinence three months after delivery. *Br J Obstet Gynaecol* 1996; 103(2):154-161.
- (2) Goldberg RP, Kwon C, Gandhi S, Atkuru LV, Sorensen M, Sand PK. Urinary incontinence among mothers of multiples: the protective effect of cesarean delivery. *Am J Obstet Gynecol* 2003; 188(6):1447-1450.
- (3) Dietz HP, Bennett MJ. The effect of childbirth on pelvic organ mobility. *Obstet Gynecol* 2003; 102(2):223-228.
- (4) Farrell SA, Allen VM, Baskett TF. Parturition and urinary incontinence in primiparas. *Obstet Gynecol* 2001; 97(3):350-356.
- (5) Allen RE, Hosker GL, Smith AR, Warrell DW. Pelvic floor damage and childbirth: a neurophysiological study. *Br J Obstet Gynaecol* 1990; 97(9):770-779.
- (6) Iosif CS, Ingemarsson I. Prevalence of stress incontinence among women delivered by elective cesarian section. *Int J Gynaecol Obstet* 1982; 20(2):87-89.
- (7) Faundes A, Guarisi T, Pinto-Neto AM. The risk of urinary incontinence of parous women who delivered only by cesarean section. *Int J Gynaecol Obstet* 2001; 72(1):41-46.
- (8) Peschers UM, Sultan AH, Jundt K, Mayer A, Drinovac V, Dimpfl T. Urinary and anal incontinence after vacuum delivery. *Eur J Obstet Gynecol Reprod Biol* 2003; 110(1):39-42.
- (9) Buchsbaum GM, Chin M, Glantz C, Guzick D. Prevalence of urinary incontinence and associated risk factors in a cohort of nuns. *Obstet Gynecol* 2002; 100(2):226-229.
- (10) Rortveit G, Daltveit AK, Hannestad YS, Hunskaar S. Vaginal delivery parameters and urinary incontinence: the Norwegian EPINCONT study. *Am J Obstet Gynecol* 2003; 189(5):1268-1274.
- (11) Persson J, Wolner-Hanssen P, Rydhstroem H. Obstetric risk factors for stress urinary incontinence: a population-based study. *Obstet Gynecol* 2000; 96(3):440-445.
- (12) MacLennan AH, Taylor AW, Wilson DH, Wilson D. The prevalence of pelvic floor disorders and their relationship to gender, age, parity and mode of delivery. *BJOG* 2000; 107(12):1460-1470.
- (13) Hvidman L, Foldspang A, Mommsen S, Nielsen JB. Postpartum urinary incontinence. *Acta Obstet Gynecol Scand* 2003; 82(6):556-563.
- (14) Wijma J, Potters AE, de Wolf BT, Tinga DJ, Aarnoudse JG. Anatomical and functional changes in the lower urinary tract following spontaneous vaginal delivery. *BJOG* 2003; 110(7):658-663.

- (15) Wijma J, Weis Potters AE, de Wolf BT, Tinga DJ, Aarnoudse JG. Anatomical and functional changes in the lower urinary tract during pregnancy. *BJOG* 2001; 108(7):726-732.
- (16) Bland JM, Altman DG. Statistical methods for assessing agreement between two methods of clinical measurement. *Lancet* 1986; 1(8476):307-310.
- (17) Versi E, Orrego G, Hardy E, Seddon G, Smith P, Anand D. Evaluation of the home pad test in the investigation of female urinary incontinence. *Br J Obstet Gynaecol* 1996; 103(2):162-167.
- (18) Lose G, Jorgensen L, Thunedborg P. 24-hour home pad weighing test versus 1-hour ward test in the assessment of mild stress incontinence. *Acta Obstet Gynecol Scand* 1989; 68(3):211-215.
- (19) van Geelen JM, Lemmens WA, Eskes TK, Martin CB, Jr. The urethral pressure profile in pregnancy and after delivery in healthy nulliparous women. *Am J Obstet Gynecol* 1982; 144(6):636-649.
- (20) Dietz HP, Steensma AB. Which women are most affected by delivery-related changes in pelvic organ mobility? *Eur J Obstet Gynecol Reprod Biol* 2003; 111(1):15-18.
- (21) King JK, Freeman RM. Is antenatal bladder neck mobility a risk factor for postpartum stress incontinence? *Br J Obstet Gynaecol* 1998; 105(12):1300-1307.
- (22) Dietz HP, Hansell NK, Grace ME, Eldridge AM, Clarke B, Martin NG. Bladder neck mobility is a heritable trait. *BJOG* 2005; 112(3):334-339.
- (23) Dietz HP, Clarke B, Herbison P. Bladder neck mobility and urethral closure pressure as predictors of genuine stress incontinence. *Int Urogynecol J Pelvic Floor Dysfunct* 2002; 13(5):289-293.
- (24) Pregazzi R, Sartore A, Bortoli P, Grimaldi E, Troiano L, Guaschino S. Perineal ultrasound evaluation of urethral angle and bladder neck mobility in women with stress urinary incontinence. *BJOG* 2002; 109(7):821-827.
- (25) Howard D, Miller JM, DeLancey JO, Ashton-Miller JA. Differential effects of cough, valsalva, and continence status on vesical neck movement. *Obstet Gynecol* 2000; 95(4):535-540.
- (26) Unterweger M, Marincek B, Gottstein-Aalame N, Debatin JF, Seifert B, Ochsenbein-Imhof N et al. Ultrafast MR imaging of the pelvic floor. *AJR Am J Roentgenol* 2001; 176(4):959-963.
- (27) Meyer S, Schreyer A, De Grandi P, Hohlfeld P. The effects of birth on urinary continence mechanisms and other pelvic-floor characteristics. *Obstet Gynecol* 1998; 92(4 Pt 1):613-618.

- (28) Woolley RJ. Benefits and risks of episiotomy: a review of the English-language literature since 1980. Part I. *Obstetrical* 1995; 50(11):806-820.
- (29) Woolley RJ. Benefits and risks of episiotomy: a review of the English-language literature since 1980. Part II. *Obstetrical* 1995; 50(11):821-835.
- (30) Sartore A, De Seta F, Maso G, Pregazzi R, Grimaldi E, Guaschino S. The effects of mediolateral episiotomy on pelvic floor function after vaginal delivery. *Obstet Gynecol* 2004; 103(4):669-673.
- (31) Keane DP, Sims TJ, Abrams P, Bailey AJ. Analysis of collagen status in premenopausal nulliparous women with genuine stress incontinence. *Br J Obstet Gynaecol* 1997; 104(9):994-998.
- (32) Jensen JK, Nielsen FR, Jr., Ostergard DR. The role of patient history in the diagnosis of urinary incontinence. *Obstet Gynecol* 1994; 83(5 Pt 2):904-910.
- (33) Frazer MI, Haylen BT, Sutherst JR. The severity of urinary incontinence in women. Comparison of subjective and objective tests. *Br J Urol* 1989; 63(1):14-15.
- (34) Ryhammer AM, Laurberg S, Djurhuus JC, Hermann AP. No relationship between subjective assessment of urinary incontinence and pad test weight gain in a random population sample of menopausal women. *J Urol* 1998; 159(3):800-803.





# CHAPTER 5

## Displacement and recovery of the vesical neck position during pregnancy and after childbirth

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## ABSTRACT

The aim of this study is (i) to describe the displacement and recovery of the vesical neck position during pregnancy and after childbirth and (ii) to discriminate between compliance of the vesical neck supporting structures with and without pelvic floor contraction.

In the present study we focussed on the biomechanical properties of the vesical neck supporting structures during pregnancy and after childbirth by calculating the compliance and the hysteresis as a result from of abdominal pressure measurements and simultaneous perineal ultrasound.

This study shows that compliance of the supporting structures remains relatively constant during pregnancy and returns to normal values 6 months after child birth. Hysteresis however showed an increase after child birth, persisting at least until 6 months post partum.

Vaginal delivery may stretch and or load beyond the physiological properties of the pelvic floor tissue and in this way may lead to irreversible changes in tissue properties which play an important role in the urethral support continence mechanism.

**Key words** Urethral support, pelvic floor, vaginal delivery, compliance and hysteresis.

## INTRODUCTION

In an overview article Gregory and Nygaard about childbirth and pelvic floor disorders describe the urinary continence mechanism depends among other factors on urethral support of both endopelvic fascia and muscles of the pelvic floor<sup>1</sup>. In this respect the biomechanical properties of the pelvic floor are of great importance, since the pelvic floor must be capable in providing sufficient back pressure for urethral closure.

The pelvic floor mainly consists of connective tissue and muscles. De Lancey discusses the normal structure of the pelvic floor and its basic function in relation to its structural biomechanics<sup>2</sup>. He stresses the important role of the endopelvic fascia which attaches the pelvic organs to the pelvic wall, in this way suspending and supporting the pelvic organs. Connective tissue undergoes significant remodelling of its components in response to a variety of factors or mechanical stresses. Pregnancy and childbirth may be among these factors.

In a review article on the muscles of the pelvic floor, Wall gives a description of muscle contribution to urethro-vesical support<sup>3</sup>. Declined muscle function due to impaired muscle integrity and neural control mechanisms directly influence this supportive mechanism. Moreover the pelvic floor muscles exhibit a constant baseline tone. By maintaining closure of the pelvic floor, the viscera can rest on this muscular shelf, keeping the ligaments from stretching. Reduction of muscle function due to pregnancy and or delivery may therefore be indirectly responsible for stretching the pelvic floor connective tissues.

In the present study we focussed on the biomechanical properties of the vesical neck supporting structures during pregnancy and after childbirth. Generally, tissue distensibility is determined not only by the elasticity of the constituent muscle and connective fibers itself but also by the geometrical arrangement of those fibers. Two parameters are in use to describe the mechanical properties of tissue: Young's modulus, or its reciprocal (the compliance), to quantify the pure elastic behaviour of tissue in which the geometrical structure of the fibers remain unaltered, and hysteresis: the failure of tissue to follow the same course during relaxation as it did during distension. The latter parameter is thought to be the result of shifts in the geometrical structure of the fibers with respect to each other, and can be

interpreted as a form of internal friction within the tissue.

Also, inertial effects involved may effect the return of the tissues to a normal position.

By the use of perineal ultrasound and simultaneous abdominal pressure measurement we were able to measure the displacement and the recovery of the vesical neck in relation to the increase of abdominal pressure during coughing and during Valsalva manoeuvre, in this way measuring its compliance during cough and during Valsalva. Also the course of the displacement could be followed during increase and decrease of the abdominal pressure, from which hysteresis could be calculated. These measurements were made throughout pregnancy and after childbirth resulting in serial measurement of the biomechanical properties of the vesical neck supporting structures during pregnancy and after childbirth.

The aim of this study is (i) to describe the displacement and recovery of the vesical neck position during pregnancy and after childbirth and (ii) to discriminate between the compliance with and without pelvic floor contraction of the levator muscles.

## METHODS

The present study is part of a prospective study on the effect of pregnancy and delivery on changes in pelvic floor mobility and urinary incontinence<sup>4,5</sup>. One hundred and seventeen women who attended the outpatient clinics of the University Hospital Groningen and the Martini Hospital Groningen enrolled for the study. The women ranged in age from 17 to 41 years (mean and median 30 years). All women were of Caucasian origin, except three who were of Mediterranean origin. All women were nulliparous and had no history of incontinence, pelvic operations or neurological disease. Written informed consent was obtained from all participating women. The study was approved by the medical ethical committees of both hospitals. For the study women were investigated three times during pregnancy, at 12-16 weeks, at 28-32 weeks and at 36-38 weeks of pregnancy. After childbirth women were investigated twice, at 6 weeks and 6 months post partum. During the study six women withdrew because of matters of inconvenience, and three had their delivery preceding the third visit. Numbers of women with available data were 98, 80 and

76 at the consecutive visits. Fifty women had complete data at all consecutive visits, these data were analysed in the current study. Bias was ruled out as women with incomplete ultrasound data did not differ from the women with complete data for those visits of which data were available. All the 50 women had spontaneous or assisted vaginal delivery. An age-matched group of 27 nulliparous non-pregnant continent women from the infertility outpatient clinic was included as a reference group. These non-pregnant controls were subjected to the same protocol, but only once.

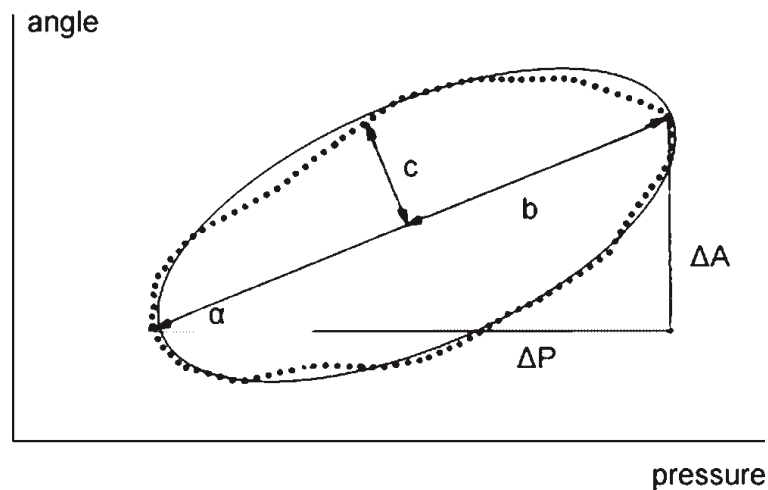
Perineal ultrasound was performed with Aloka 600 equipment with a 3.5 MHz convex transducer and the woman in the lithotomy position. Bladder volumes as calculated by scan at the three subsequent measurements varied from 100-300 ml, the mean (sd) being 238 (51), 254 (60) and 222 (54). No significant differences at the subsequent measurements. The position of the urethro-vesical junction was recorded continuously during coughing and during the Valsalva maneuver (figure 1), standardized as described before<sup>4</sup>, for coughing over 70 cm H<sub>2</sub>O and for Valsalva over 50 cm H<sub>2</sub>O was needed before measurements were made. Simultaneous abdominal pressure changes were recorded by the insertion of a microtip pressure transducer (Gaeltec) high in the posterior fornix of the vagina. All scans were made by the first and second author. Inter and intra observer variability was less than 5%. A more extensive description of the methods can be found in a previous article<sup>4</sup>.

During the Valsalva manoeuvre and during coughing a set of data of displacement of the urethro-vesical junction and the change in abdominal pressure was obtained, as shown in figure 2, showing the relationship between displacement of the urethro-vesical junction and abdominal pressure, i.e. the stiffness of the vesical neck supporting structures. In physiology often the reciprocal of the stiffness is used, the compliance: the increase in length per unit force applied. In more dimensional structures compliance is also defined as the increase in extension per unit pressure. As a parameter directly related to extension of the pelvic floor we used the change in the urethro-vesical junction angle under application of pressure. Pressure was generated by both letting the subjects perform a Valsalva manoeuvre and during voluntary coughing. Since the change in urethro-vesical junction angle is independent of the anatomical dimensions, the resulting parameter, change in angle divided by the pressure generated, can be considered as the specific compliance with as

Figure 1 consists of two B-mode echocardiographic images of the left ventricle. The left image shows a normal heart with a normal septal thickness (S) and a normal angle ( $\beta_1$ ) between the septum and the posterior wall. The right image shows a heart with a thickened septum (S) and a larger angle ( $\beta_2$ ) between the septum and the posterior wall. Both images include a scale bar at the bottom and technical data on the right side.

Using SPSS for windows, all continuous variables were tested for normal distribution using the Kolmogorov-Smirnov test; no continuous variables were assigned to differ from the normal distribution. For comparison between groups of continuous variables the t-test for equality of means was used.

Figure 2: Calculation of compliance and hysteresis



Compliance, the displacement/pressure coefficient, expressed as the displacement of the urethro-vesical junction in degrees per abdominal pressure change in cm H<sub>2</sub>O,  $\Delta A/\Delta P$ .  
Hysteresis, the failure of tissue to follow the same course during relaxation as it did during distension, expressed as the area of the curve, estimated as area of an the standardized ellipse  $\pi \cdot 1 \cdot c/b$ .

## RESULTS

During pregnancy the *compliance during coughing* remains practically unchanged. After childbirth the compliance during coughing increases significantly. At 6 months post partum the compliance during coughing differs no longer from coughing compliance in the control group, figure 3.

For the *compliance during Valsalva* there is the same trend during pregnancy, no significant changes. After childbirth, compliance during Valsalva increases significantly. After 6 months the compliance during Valsalva has returned to the value of the control group, figure 4.

The "*muscle compliance*", as calculated from compliance during coughing and during Valsalva is not influenced by pregnancy. After childbirth "*muscle compliance*" significantly increases. 6 Months after childbirth "*muscle compliance*" is comparable to the value as found in the control group, figure 5.

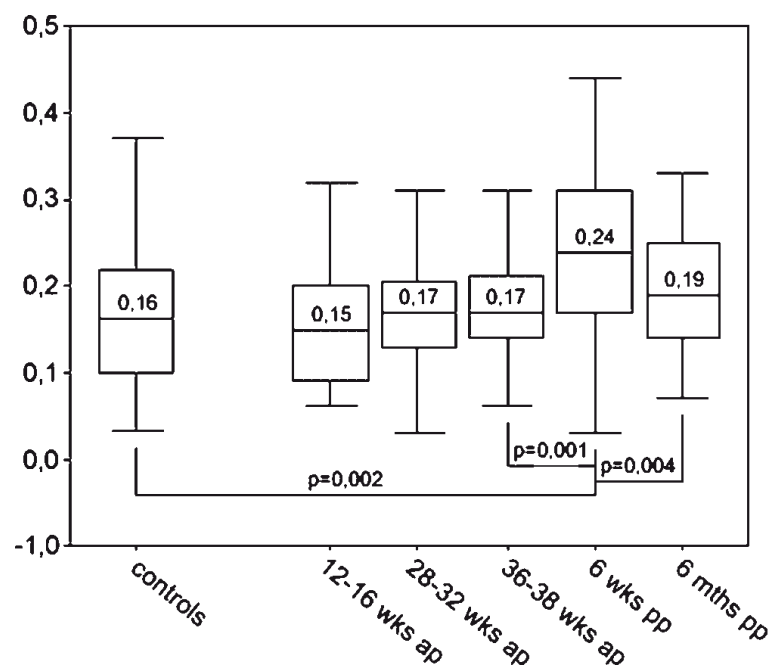
Hysteresis as calculated during the Valsalva manoeuvre (figure 6) and during coughing (figure 7) is not influenced by pregnancy. After childbirth there is a significant rise, persisting at least until 6 months post partum.

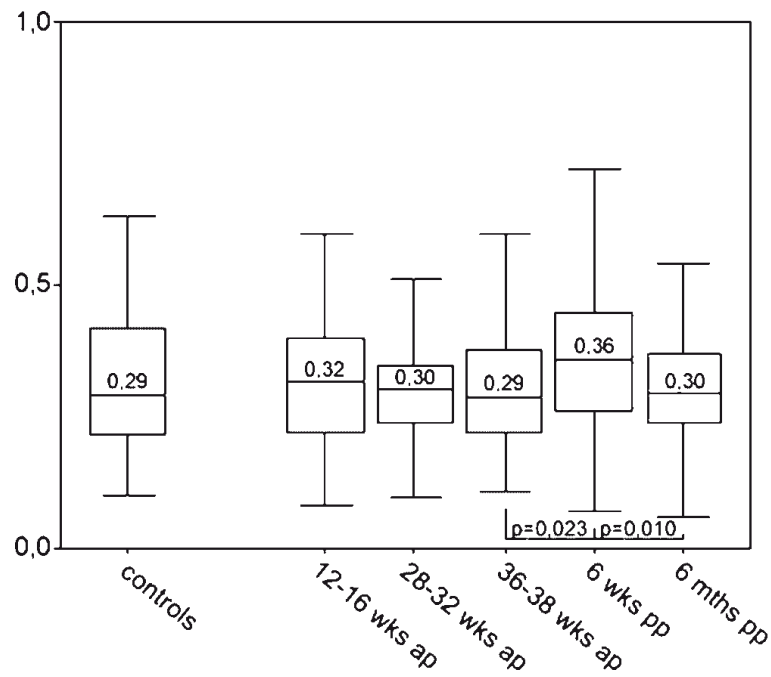
## DISCUSSION

This study shows that compliance of the vesical neck supporting structures remains relatively constant during pregnancy and returns to normal values 6 months after child birth. Hysteresis however showed an increase after childbirth, persisting at least until 6 months post partum.

Stiffness, or its more mechanical denomination elasticity, of the pelvic floor is an important biomechanical property. It is usually defined as the force to be applied necessary to achieve a predetermined percentage or fraction increase in length. In physiology often the reciprocal of the elasticity is used, the compliance: the increase in length per unit force applied. In more dimensional structures compliance is also defined as the increase in extension per unit pressure.

Figure 3: Compliance during coughing



**Figure 4:** Compliance during Valsalva

In general, deformation under load for tissue is seldomly linear. To quantify such deformation we decomposed deformation into elasticity, approximated by a linear model and hysteresis, estimated by a non linear model as shown in figure 1.

The compliance of the vesical neck supporting structures seems to be significantly affected by childbirth as measured by the increased compliance 6 weeks after delivery. 6 Months after delivery compliance has normal values, comparable with an age matched nulliparous non pregnant control group. Our data suggest that childbirth in primiparous women only affects this dynamic pelvic floor tissue properties temporary. This holds for the pelvic floor tissue during Valsalva manoeuvre as well as during coughing.



Figure 5: Muscle compliance

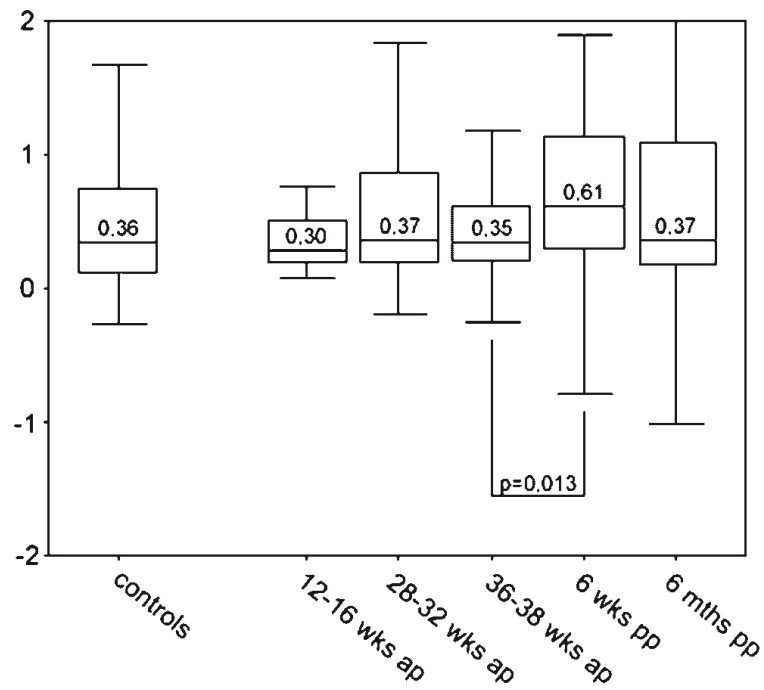
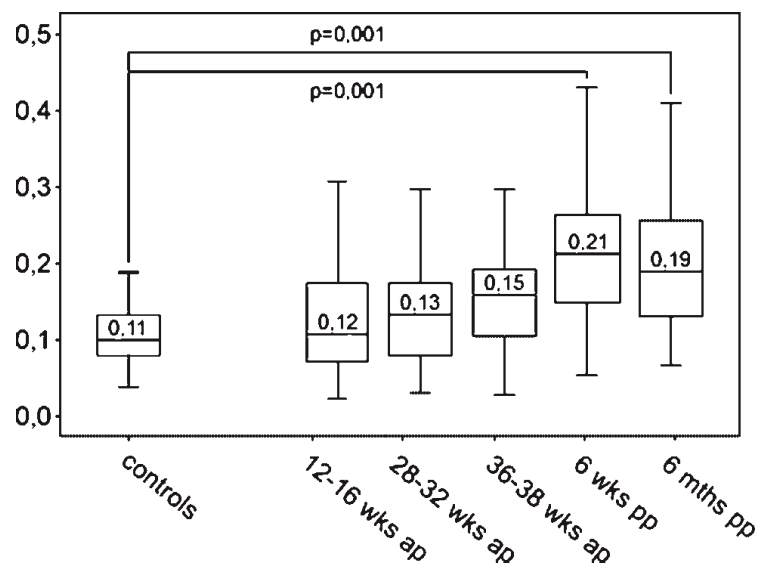
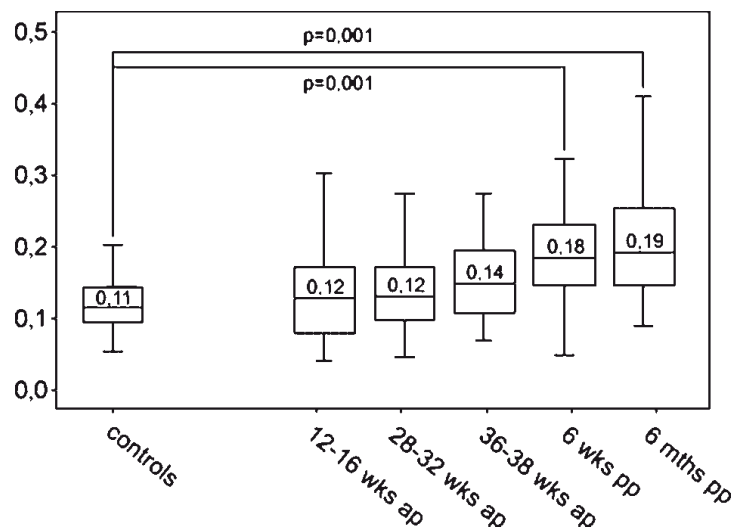


Figure 6: Hysteresis during Valsalva



The increase in hysteresis however seems to be permanent. Since hysteresis, the phenomenon that tissue does not follow the same course during distention as during relaxation, depends on shifts of the constituent fibers with respect to each other, an increase in hysteresis means that the geometrical structure becomes looser. Both compliance and hysteresis contribute to the stiffness of the pelvic floor and our data suggest that a decrease in stiffness of the pelvic floor after child birth is mainly due to a more loosely coupling of the various fibers in the pelvic floor tissue to each other. Reduced fascial strength and decreased muscle tone tend to make the pelvic floor more flaccid so that the necessary back pressure cannot be produced leading to hypermobility of the Urethro Vesical Junction, significantly related with genuine urinary stress incontinence<sup>6</sup>.

Figure 7: Hysteresis during coughing



The compliance of the pelvic floor in vivo is determined by both muscle tone and the properties of the fascia. During the Valsalva manoeuvre no additional muscle tone originating from the pelvic floor muscles was supposed to be present. Little contraction of the pelvic floor muscles cannot be ruled out because straining induces levator ani muscle contraction, more pronounced in sudden straining than in slow sustained straining<sup>7</sup>. Moreover women do not always find it possible to relax their muscles during Valsalva's maneuver, especially if they are concerned about

loosing gas. Therefore we instructed patients emphatic to avoid pelvic floor muscle contraction. This is described to be effective to relax pelvic floor muscles as measured by reduced EMG activity<sup>8</sup>. During coughing a reflex contraction of the suburethral pelvic floor muscles occurs<sup>9</sup>. In this way we have two measurements of compliance under different muscle tone, which allow us to focus on the contribution of muscle activity to the pelvic floor compliance.

Increased post partum Valsalva compliance means less resistance to abdominal pressure rises and hypermobility of the viscera and its supported organs in the post partum period. In our study Valsalva compliance returns to normal values after 6 months, indicating normal dynamic function of the fascia at least during slow abdominal pressure rises, such as Valsalva.

Norton reviews on the role of fascia and ligaments<sup>10</sup>. Connective tissue of the pelvic floor mainly consists of type 3 collagen. The collagen fibres are randomly layered and give the pelvic viscera its elastic properties. Fascia are resistant to great forces. It is thought that as the tissue is stretched the crimp gradually disappears by straightening the collagen fibres. Stretching or loading beyond this point may lead to irreversible damage<sup>11</sup>. Stress relaxation is thought to be an accommodation effect of fascia to pregnancy and as a preparation to childbirth. Cervical ripening after prostaglandins is associated with collagen fiber re-arrangement and increase of ground substance<sup>12</sup>. It is likely that these alterations take place in all collagen tissue. This may contribute to lack of urethro vesical support during pregnancy, thus leading to (temporary) hypermobility, a well known risk factor for urinary incontinence.

Tissue consisting of structures each having different elastic properties has a total elasticity which can be calculated as the sum of the elasticity's of the components. By converting the measured compliances to elasticity (the reciprocal value) and subtracting the values obtained during coughing and those obtained at the Valsalva manoeuvre we calculated the effect of contraction of the pelvic floor muscles to the pelvic floor stiffness, using the following estimate:

$$1/\text{compliance (contracted muscles)} = 1/\text{compliance Cough} - 1/\text{compliance Valsalva}$$

The method we used to estimate the compliance of the contracted pelvic floor muscles may have its limitations. Undoubtedly there exists a remaining muscle tone

during the Valsalva manoeuvre<sup>7</sup>. It should be kept in mind that the compliance values measured during the Valsalva manoeuvre cannot be completely ascribed to the mechanical properties of endopelvic fascia alone, but also reflect some muscle tone, thus effecting the outcome in our estimation. In conclusion the separation of compliance between fascia and relaxed muscle on one hand and fascia and contracted muscle on the other hand may not be complete. However, a complete separation cannot be realized in *in vivo* measurements. Moreover other effects may explain these differences, such as muscle integrity and neural control mechanisms. To obtain quantitative insight in the course of the mechanical properties of the pelvic floor during pregnancy and after childbirth *in vivo* measurements are necessary and the method used in this study may therefore be a good approximation. For convenience the estimated compliance of the contracted muscles is referred to as "*Muscular compliance*".

"*Muscular compliance*" is, similar to fascial compliance during Valsalva, only temporary affected. Pelvic muscle groups support structures which penetrate the urogenital hernia<sup>13</sup>. During stressful periods of increased intra abdominal pressure, such as coughing these muscle groups provide support by reflex contraction. The passing fetal head may lead to direct muscular or indirect neurological injury due to (over)stretching. This may lead to hypermobility of the urethro vesical junction and to urinary incontinence. From our results we conclude that muscular function itself during coughing is not permanently significantly affected by pregnancy and childbirth.

Barbic described increased compliance of the pelvic floor during coughing as a distinguishing mark in women with stress urinary incontinence<sup>14</sup>. He found a greater compliance in his study group of parous women with stress urinary incontinence (SUI) as compared to a control group without SUI. Measurements were done during coughing in non pregnant parous women, no Valsalva measurements are reported. Howard reported on stiffness of the pelvic floor both during coughing and during Valsalva<sup>15</sup>. For coughing he found a decrease of stiffness in stress incontinent primiparous women as compared to nulliparous continent women. For Valsalva no difference between these groups was found. Our results do not indicate permanent increase of compliance due to childbirth for the study group, nor for coughing, nor for Valsalva. The fact that we have not found significant differences, does not mean

that there were not individual women who lost support. It should be emphasized that these women may have permanent increased compliance and may be the ones who develop SUI directly or later in life time.

Calculation of pelvic muscle compliance is not described before. The pelvic floor muscular impact on urinary stress incontinence is qualitatively studied by measuring the cough leak point pressure (CLPP) and Valsalva leak point pressure (VLPP)<sup>7,16</sup>. A greater CLPP than VLPP in an incontinent woman indicates the presence of an effective contraction of the suburethral tissue during coughing. Women with grade III SUI had lower VLPP than CLPP, indicating more intrinsic sphincter deficiency (ISD). In these women less hypermobility of the urethro vesical junction was found than in women with a lower CLPP. From their study on CLPP and VLPP Pescher et al.<sup>17</sup> conclude that these provocations result in a different reaction of the pelvic floor, possibly due to an involuntary isotonic or isometric contraction of the pelvic floor muscles stabilising the pelvic floor. Another way of measuring the pelvic floor muscular continence component is imaging of voluntary pelvic floor contractions<sup>18</sup>. The latter gives information on voluntary contraction but does not measure the function of the pelvic floor during the provocative moment of coughing, therefore the clinical use of it is probably limited.

In our study we described changes in the dynamics of urethral support structures during coughing and during Valsalva throughout pregnancy and up to 6 months after childbirth. We found that the compliance and of the pelvic floor is only temporary affected by the process of childbearing, both during coughing as well as during Valsalva. We described a theoretical approximation of muscular and fascia contribution to compliance of the pelvic floor. Both muscular tissue and connective tissue seem to be resistant to the process of childbirth as concluded from the values for both compliances after six months, being not different from the measurements in non pregnant controls. Hysteresis however showed an increase after child birth at least persisting until six months post partum, showing that delivery may stretch and or load beyond the physiological properties of the pelvic floor tissue and in this way may lead to irreversible changes in tissue properties which play an important role in the urethral support continence mechanism.

## REFERENCE LIST

- (1) Gregory WT, Nygaard I. Childbirth and pelvic floor disorders. *Clin Obstet Gynecol* 2004; 47(2):394-403.
- (2) DeLancey JO. Anatomy and biomechanics of genital prolapse. *Clin Obstet Gynecol* 1993; 36(4):897-909.
- (3) Wall LL. The muscles of the pelvic floor. *Clin Obstet Gynecol* 1993; 36(4):910-925.
- (4) Wijma J, Weis Potters AE, de Wolf BT, Tinga DJ, Aarnoudse JG. Anatomical and functional changes in the lower urinary tract during pregnancy. *BJOG* 2001; 108(7):726-732.
- (5) Wijma J, Potters AE, de Wolf BT, Tinga DJ, Aarnoudse JG. Anatomical and functional changes in the lower urinary tract following spontaneous vaginal delivery. *BJOG* 2003; 110(7):658-663.
- (6) DeLancey JO. Structural support of the urethra as it relates to stress urinary incontinence: the hammock hypothesis. *Am J Obstet Gynecol* 1994; 170(6):1713-1720.
- (7) Shafik A, El Sibai O, Shafik AA, Ahmed I. Effect of straining on perineal muscles and their role in perineal support: identification of the straining-perineal reflex. *J Surg Res* 2003; 112(2):162-167.
- (8) Giannantoni A, Di Stasi SM, Cucchi A, Mearini E, Bini V, Porena M. Pelvic floor muscle behavior during Valsalva leak point pressure measurement in males and females affected by stress urinary incontinence. *J Urol* 2003; 170(2 Pt 1):485-489.
- (9) Amarenco G, Ismael SS, Lagauche D, Raibaut P, Rene-Corail P, Wolff N et al. Cough anal reflex: strict relationship between intravesical pressure and pelvic floor muscle electromyographic activity during cough. Urodynamic and electrophysiological study. *J Urol* 2005; 173(1):149-152.
- (10) Norton PA. Pelvic floor disorders: the role of fascia and ligaments. *Clin Obstet Gynecol* 1993; 36(4):926-938.
- (11) Landon CR, Crofts CE, Smith ARB, Trowbridge EA. Mechanical properties of fascia during pregnancy: a possible factor in the development of stress incontinence of urine. *Contemp Rev Obstet Gynaecol* 1990; 2:40-46.
- (12) Uldbjerg N, Ekman G, Malmstrom A, Olsson K, Ulmsten U. Ripening of the human uterine cervix related to changes in collagen, glycosaminoglycans, and collagenolytic activity. *Am J Obstet Gynecol* 1983; 147(6):662-666.
- (13) Lawson JO. Pelvic anatomy. I. Pelvic floor muscles. *Ann R Coll Surg Engl* 1974; 54(5):244-252.

- (14) Barbic M. Compliance of the bladder neck supporting structures: importance of activity pattern of levator ani muscle and content of elastic fibers of endopelvic fascia. *Neurourology and urodynamics* 2003; 22(4):269-276.
- (15) Howard D, Miller JM, DeLancey JO, Ashton-Miller JA. Differential effects of cough, valsalva, and continence status on vesical neck movement. *Obstet Gynecol* 2000; 95(4):535-540.
- (16) Kuo HC. Videourodynamic analysis of the relationship of Valsalva and cough leak point pressures in women with stress urinary incontinence. *Urology* 2003; 61(3):544-548.
- (17) Peschers UM, Jundt K, Dimpfl T. Differences between cough and Valsalva leak-point pressure in stress incontinent women. *Neurourol Urodyn* 2000; 19(6):677-681.
- (18) Constantinou CE, Hvistendahl G, Ryhammer A, Nagel LL, Djurhuus JC. Determining the displacement of the pelvic floor and pelvic organs during voluntary contractions using magnetic resonance imaging in younger and older women. *BJU Int* 2002; 90(4):408-414.



# CHAPTER 6

## The diagnostic strength of the 24-hour pad test for self reported symptoms of urinary incontinence in pregnancy and after childbirth

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**ABSTRACT**

The clinical impact of incontinence in pregnancy and after childbirth is growing since some studies report the efficacy of physiotherapy in pregnancy and since obstetric choices are supposed to have significant impact on post reproductive urinary function [1]. Thus the need for objective measurement of urinary incontinence in pregnancy is growing. Data on pad testing in pregnancy are lacking. We assessed the clinical relevance of the 24 hour pad test during pregnancy and after childbirth, compared with data on self reported symptoms of urinary incontinence and visual analogue score (VAS). According to the ROC curve the diagnostic value of pad testing for measuring (severity of) self reported incontinence during pregnancy is not of clinical relevance. However, for the purposes of research, pad tests, combined with subjective/qualitative considerations, play a critical role in allowing comparisons across studies, quantifying the amount of urine loss, and establishing a measure of severity.

**Keywords** pad test, urinary incontinence, pregnancy, childbirth and diagnostic strength

***Brief summary***

Pad testing did not show to have high sensitivity and specificity for self reported urinary incontinence in pregnancy and after childbirth. Confusion about the diagnosis remains.

## INTRODUCTION

Incontinence is reported frequently in pregnancy and after childbirth[2,3,4]. It has been suggested that urinary incontinence in pregnancy is a predictor of the chance to develop post partum urinary incontinence[5]. In that respect prevention such as physiotherapy during pregnancy is advised in women with positive symptoms of urinary incontinence in pregnancy [6,7,8,9]. Based on questionnaires on symptoms of post partum urinary incontinence Goldberg et al found a strong protective effect of cesarean delivery against the development of post partum urinary incontinence and highlighted the impact of obstetric choices on post reproductive urinary function[1]. When the possible consequences of the fact that a pregnant women reports urinary incontinence grow, such as an advise for preventive physiotherapy or an advice regarding the mode of delivery , there is a greater need for objectivity in diagnosing the problem. Pad testing yields an objective measurement of fluid loss over a certain period. In non pregnant women the diagnostic value of pad testing for self reporting of symptoms of urinary incontinence has been questioned[10,11]. These data are lacking for pregnant women. Most common used types of pad tests are the one-hour and the 24 hour test[12,13,14,15]. The 24-hour pad test is almost certainly more representative to the patients' day-to-day experiences and is more likely to correlate with self reported symptoms[16]. 24 Hour pad testing has been studied and described exclusively in non pregnant women. In this study we focussed on the diagnostic strength of pad testing to measure (the severity of) urinary incontinence in pregnancy and after childbirth. The aim of this study was (i) to describe pad weight gain as measured by the 24-hour pad test in a cohort of pregnant women and (ii) to assess the clinical usefulness of the 24 hour pad test in pregnancy and after childbirth in terms of the relationship between objective urine loss and the self reported symptoms of urinary incontinence.

## METHODS

One hundred and seventeen women who attended the outpatient clinics of the University Hospital Groningen and the Martini Hospital Groningen enrolled for the study, mean age 30 years (range 17-41). All women were of Caucasian origin, except three who were of Mediterranean origin. All women were nulliparous and had no

history of incontinence, pelvic operations or neurological disease. Written informed consent was obtained from all participating women. The study was approved by the medical ethical committees of both hospitals. For this study the women were investigated at 28-32 weeks and at 36-38 weeks of pregnancy, and six weeks and six months post partum.

At each visit all women completed a questionnaire and a visual analogue score (VAS) on symptoms of urinary incontinence. Complaints of more than five on the VAS scale 0-10 were defined as severe complaints. Women were asked to classify their incontinence as mainly: 1) stress urinary incontinence, involuntary leakage on effort or exertion, or on sneezing or coughing, 2) urge urinary incontinence, involuntary leakage accompanied by or immediately preceded by a strong desire to void[17]. Three pads were packed, each in a plastic bag and weighed by the investigators before and after use. The women received a written instruction and were free to wear one, two or three pads. It was emphasised that the bag should be closed carefully every time a pad was changed to prevent evaporation. If the bags were open or less than three pads were returned the test was excluded from evaluation. Pads were given to all women, to be worn for 24 hours preceding their appointment. The outcome of the 24-hour pad test was recorded as the weight gain as measured by a verified spring balance. Weighing was done by the first or second author, within three days after the pad test was carried out. According to the literature pads were assigned as wet if the total weight gain per 24 hour was  $\geq 9$  gram[11].

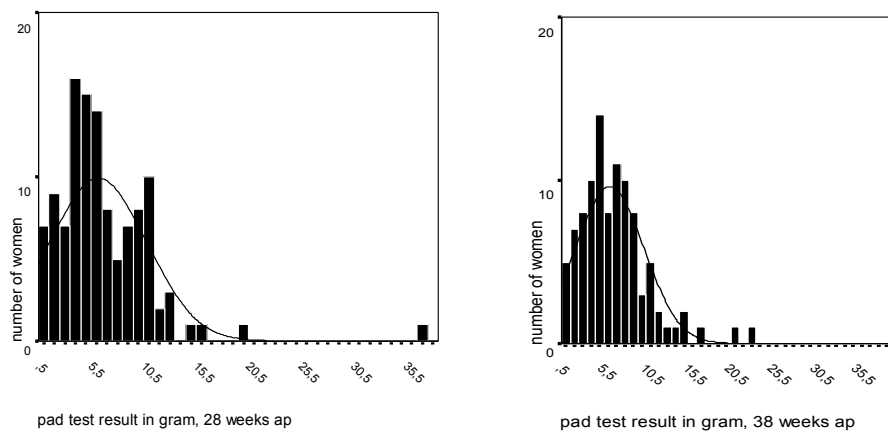
## STATISTICAL ANALYSIS

Pad test results have non parametric distribution. For continuous variables non parametric tests are used. Numeric data are analysed by cross tabulation, chi-square test and risk analysis. Pearson correlation test was used to identify significant relationships between variables. Data are presented as median or numbers.

## RESULTS

### *Pregnancy, pad test*

At 28 weeks of pregnancy 115/117 patients (98%) returned their pads according to the protocol. The median weight gain was 5 gram (range 0-36). At 38 weeks data were available from 98 women (84%). Two patients had withdrawn from the study because of inconvenience, while 17 pads were not, or were not according to the protocol returned. At this stage of pregnancy the median weight gain was also 5 gram (range 0-22). Distributions of pad test weight gain are given in figure 1.



**Figure 1:** Distribution of 24 hour pad test results at 28 and 38 weeks of pregnancy

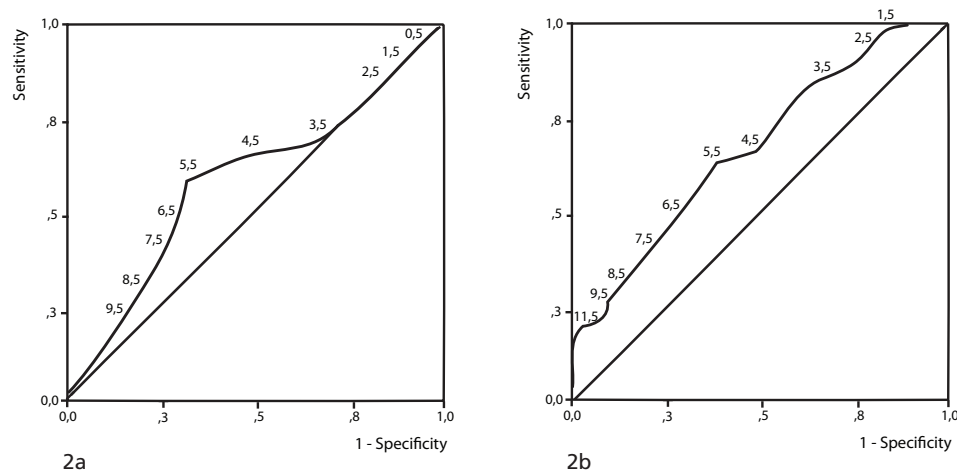
Pad test results at 28 and 38 weeks of pregnancy were related ( $r = 0.452$ ,  $p < 0.0001$ ). Twenty-seven out of 115 (23%) and 17 out of 98 pads (17%) were wet at 28 and 38 weeks of pregnancy respectively and again results at 28 and 38 weeks were related ( $r = 0.317$ ,  $p < 0.001$ ).

### *Pregnancy, questionnaire*

At 28 weeks of pregnancy 35/117 women (30%) reported incontinence, (stress 28/35 and urge 7/35) and at 38 weeks 40/115 (35%) did (stress 33/40 (82%) and urge 7/40 (8%)). Reported incontinence at 28 and 38 weeks of pregnancy was related ( $r = 0.55$ ,  $p < 0.001$ ). Severe complaints of incontinence were reported in 17/117 (15%) and 22/115 cases (19%), also related ( $r = 0.482$ ,  $p < 0.001$ ).

### *Pregnancy, pad test and questionnaire*

Women with self reported incontinence at 28 and 38 weeks of pregnancy had a median pad weight gain of 6.0 gram (range 0-36) and 6.0 gram (range 0-22) respectively while women without self reported incontinence had a pad test result of 4.0 gram (range 0-22) and 4.0 gram (range 0-22) respectively, showing no differences between these groups of women. To evaluate the diagnostic value of the pad test for measuring self reported incontinence the sensitivity and specificity for several cut off levels for the pad test was calculated, graphically known as the ROC-curve. The ROC curve for pad tests and self reported incontinence at 28 weeks of pregnancy did not differ from the reference line (area 0.575 versus 0.5,  $p=0.207$ ), showing a non-diagnostic test, figure 2a. At 38 weeks of pregnancy the ROC curve showed a significant difference from the reference line (area 0.663 versus 0.5,  $p=0.008$ ), the optimum cut off point at 5.5 gram with a sensitivity of 0.629 and a specificity of 0.619, figure 2b. When stratified for stress incontinence or for severe symptoms of urinary incontinence the ROC curve did not improve.

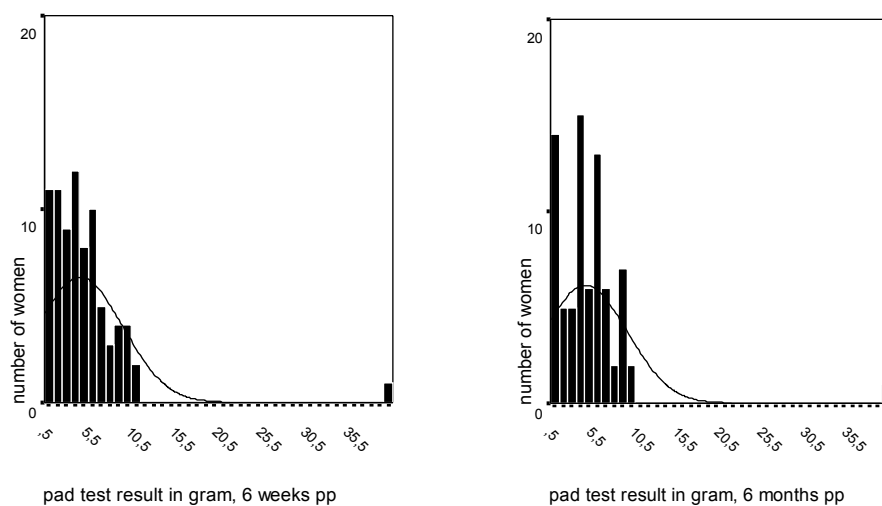


**Figure 2:** ROC curve, the diagnostic value of the pad test at different cut off levels (g/24h) for measuring self reported incontinence at 28 and 38 weeks Should refer to figure 2a and 2b, by introducing : Fig. 2 ROC curve, the diagnostic value of the pad test at different cut off levels (g/24h) for measuring self reported incontinence at 28 (2a) and 38 weeks(2b)

### *Puerperium, pad test*

At 6 weeks post partum 80/117 patients (68%) returned their pads according to the protocol. The median weight gain was 3 gram (range 0-40). At 6 months post partum pad data were available from 76 patients (65%). Six patients had withdrawn

from the study because of inconvenience, 3 patients had their delivery preterm, 32 pads were not, or were not according to the protocol returned. These women did not want to participate in the pad study anymore or did not return their pads according to the protocol. The women that withdrew from the pad test study were asked to fill up the questionnaires on symptoms of urinary incontinence to check for bias. The women did not differ for self reported symptoms on urinary incontinence from the women that continued the study on pads. In the study group the median weight gain was again 3 gram (range 0-40). Distributions of pad test weight gain are given in figure 3. Pad test results at 6 weeks and 6 months postpartum were related ( $r = 0.792$ ,  $p < 0.0001$ ). Seven out of 80 (9%) and 3 out of 76 pads (4%) were wet at 6 weeks and 6 months post partum respectively, and again the results at 28 and 38 weeks were related ( $r = 0.320$ ,  $p = 0.017$ ).



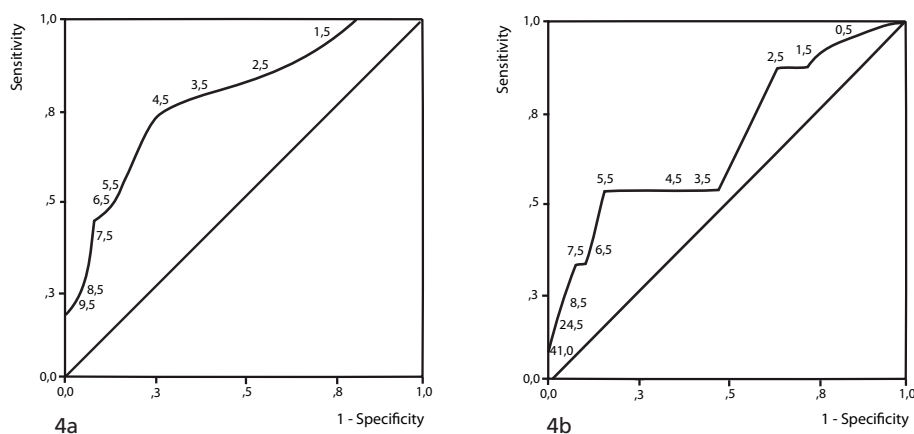
**Figure 3:** Distribution of 24 hour pad test results at 6 weeks and 6 months post partum

#### *Puerperium, questionnaire*

At 6 weeks postpartum 21/115 women (18%) reported incontinence (stress 16/21 and urge 5/21) while at 6 months postpartum 16/109 (15%) did (stress 12/16 and urge 4/16). Reported incontinence at 6 weeks and 6 months postpartum is related ( $r = 0.61$ ,  $p < 0.001$ ). Severe complaints of incontinence was reported in 15/115 (13%) and 11/109 cases (10%), also related ( $r = 0.691$ ,  $p < 0.001$ ).

### *Puerperium, pad test and questionnaire*

Women with self reported incontinence at 6 weeks and 6 months postpartum had a median pad weight gain of 5.5 (range 0-40) and 6.0 gram (range 0-40) respectively, women without self reported incontinence had a pad test result of 3.0 gram (range 0-16) and 3.0 gram (range 0-9), showing a differences between these groups of women,  $p=0.01$  and  $p=0.045$ , respectively. The ROC curve for pad tests and self reported incontinence at 6 weeks post differed from the reference line (area 0.767 versus 0.5,  $p=0.001$ ), the optimum cut off point at 4.5 gram and the sensitivity of 0.722 and specificity of 0.742, figure 4a. At 6 months post partum the ROC curve shows also a significant difference from the reference line (area 0.666 versus 0.5,  $p=0.047$ ), the optimum cut off point at 5.5 gram and the sensitivity of 0.629 and specificity of 0.619, figure 4b. Again, stratifying for stress incontinence or for severe symptoms of urinary incontinence, the ROC curve did not improve.



**Figure 4:** ROC curve, the diagnostic value of the pad test at different cut off levels (g/24h) for measuring self reported incontinence at 6 weeks and 6 months post partum should refer to figure 4a and 4b, by introducing: Fig.4 ROC curve, the diagnostic value of the pad test at different cut off levels (g/24h) for measuring self reported incontinence at 6 weeks (4a) and 6 months post partum (4b)

## **DISCUSSION**

In this study we focussed on the use of pad testing in order to investigate its prognostic value for objectively measuring (the severity of) self reported urinary incontinence during pregnancy and after childbirth. The clinical impact of incontinence in pregnancy and after childbirth is growing since some studies report the efficacy of physiotherapy in pregnancy and since obstetric choices are supposed

to have significant impact on post reproductive urinary function[1]. This growing impact requires objective measurement. In a meta analysis the symptom of stress incontinence was 91% sensitive but only 51% specific for detecting genuine stress urinary incontinence as defined by the International Continence Society, based on history and urodynamic testing[18]. Because of pregnancy and because we are interested in an instrument for screening it is obvious that urodynamic testing cannot be the instrument of choice. Pad testing is an objective, simple and non invasive instrument capable of measuring fluid loss in a certain period. First of all we need to define normal values, data which describe the results of pad testing in a cohort of pregnant women without a history of incontinence before pregnancy. Secondly we need comparison with the criterion standard in pregnancy, patients' history.

The median weight gain in the 24-hour pad test in pregnancy as reported in our study is in accordance with results reported in a group of non-pregnant, not incontinent, premenopausal women, 2.6 – 7.0 gram, with an upper confidence limit of 5.5 – 8 gram[16,19]. In postmenopausal continent women a much lower weight gain of 0.3 gram is reported[20]. When compared to the premenopausal women the state of pregnancy does not lead to a higher weight gain in the 24 hour pad test, nor does the puerperal state.

It is remarkable that the group of controls as referred to had similar pad test results but did not report incontinence, whereas in our pregnant group 30% (28 weeks of pregnancy) and 35% (38 weeks of pregnancy) of the women did. With the same weight gain in pregnancy women report more incontinence than in the sample of non pregnant women. It seems therefore that not the amount of weight gain in the pad test but the pregnancy state itself is more discriminating for the chance that a woman qualifies herself as incontinent.

In our study during pregnancy pad test results had only limited diagnostic value for self reporting of incontinence. In their review article Ryhammer et al. stated that "incontinence is a complex condition in which differences in the individual patients' personal characteristics influence the perception of leakage and the identification of the problem"[21]. Pregnancy seems to modulate this perception in such a way that it cannot be measured by pad tests. As pad testing did not show to have high sensitivity and specificity for self reported urinary incontinence in pregnancy



and after childbirth there remains confusion about the accurate diagnosis. This becomes important in deciding on management options such as offering preventive physiotherapy in selected cases or strategies which influence the mode of delivery.

After childbirth the median weight gain is also in accordance with results in non pregnant continent women. In our study group 15 to 18% of the women report positive for symptoms of incontinence. Just like in pregnancy the pad test result has a significant value for testing self reporting incontinence, but again low figures for sensitivity and specificity. Like us Morkved and Bo[22] reported discrepancy between self-reported symptoms and stress urinary incontinence assessed by their (short) pad test, 8 weeks after delivery. To assign a woman to an intervention one needs a higher specificity which according to the curves as shown, will rapidly lead to lower sensitivity. Depending on the chosen intervention this may or may not be accepted.

The calculation of the diagnostic strength of our 24 hour pad test was made with the self reporting of symptoms of urinary incontinence as the gold standard. At 28 weeks the pad test failed to capture 8 subjects who stated they were wet; at 38 weeks this was higher. Such results are possibly related to the high threshold for definition of incontinence in the women with some leaking < 9 gm describing some leakage. The rationale for using a high cut off is established in both men and women, but subjects themselves may perceive this a severe incontinence. It is possible that pregnant and post delivery women perceive leakage differently than their non-pregnant counterparts.

When adding severity of symptoms to the gold standard, as reported by visual analogue scores, the diagnostic strength of the pad test did not improve.

In general practice, questioning about incontinence will provide the clinician with adequate information on the presence, absence or severity of incontinence from a patient perspective and cumbersome pad tests are unnecessary. In a review article on questionnaires for women with pelvic floor disorders Barber concludes that measuring symptom severity and quality of life changes in women with pelvic floor disorders is an important part of the evaluation and treatment of women and may be the only practical way to clinically assess symptoms[23]. However, for the purposes of research, pad tests, combined with subjective/qualitative considerations, play a

critical role in allowing comparisons across studies, quantifying the amount of urine loss, and establishing a measure of severity. Indeed the ICS standards for research strongly recommend the pad test as one measure in all incontinence research. The fact that the pad test results and patient reported incontinence were not strongly correlated illustrates the importance of both quantitative and qualitative measures when considered an intervention trial with pelvic floor muscle exercises, for example.

From our study we conclude that pad testing measures fluid loss over a certain period, but does not quantify self reported symptoms of urinary incontinence. Both measurements are of interest, but cannot replace each other. Stressing of the pelvic floor by pregnancy and childbirth modulates the sensation of urinary leakage in such a way that women in this state do report symptoms of urinary incontinence more frequently than nulliparous premenopausal women do.

## REFERENCE LIST

- (1) Goldberg RP, Kwon C, Gandhi S, Atkuru LV, Sorensen M, and Sand PK (2003) Urinary incontinence among mothers of multiples: the protective effect of cesarean delivery. *Am J Obstet Gynecol* 188:1447-50
- (2) Francis WJ (1960) The onset of stress incontinence. *J Obstet Gynaecol Br Emp* 67:899-903
- (3) Burgio KL, Locher JL, Zyczynski H, Hardin JM, and Singh K (1996) Urinary incontinence during pregnancy in a racially mixed sample: characteristics and predisposing factors. *Int Urogynecol J Pelvic Floor Dysfunct* 7:69-73
- (4) Stanton SL, Kerr-Wilson R, and Harris VG (1980) The incidence of urological symptoms in normal pregnancy. *Br J Obstet Gynaecol* 87:897-900
- (5) King JK and Freeman RM (1998) Is antenatal bladder neck mobility a risk factor for postpartum stress incontinence? *Br J Obstet Gynaecol* 105:1300-7
- (6) Sampselle CM, Miller JM, Mims BL, DeLancey JO, Ashton-Miller JA, and Antonakos CL (1998) Effect of pelvic muscle exercise on transient incontinence during pregnancy and after birth. *Obstet Gynecol* 91:406-12
- (7) Reilly ET (2002) Prevention of postpartum stress incontinence in primigravidae with increased bladder neck mobility: a randomised controlled trial of antenatal pelvic floor exercises. *BJOG* 109:68-76

- (8) Chiarelli P and Campbell E (1997) Incontinence during pregnancy. Prevalence and opportunities for continence promotion. *Aust N Z J Obstet Gynaecol* 37:66-73
- (9) Wilson PD, Herbison RM, and Herbison GP (1996) Obstetric practice and the prevalence of urinary incontinence three months after delivery. *Br J Obstet Gynaecol* 103:154-61
- (10) Versi E and Cardozo LD (1986) Perineal pad weighing versus videographic analysis in genuine stress incontinence. *Br J Obstet Gynaecol* 93:364-6
- (11) Ryhammer AM, Laurberg S, Djurhuus JC, and Hermann AP (1998) No relationship between subjective assessment of urinary incontinence and pad test weight gain in a random population sample of menopausal women. *J Urol* 159:800-3
- (12) Matharu GGSAR (2004) Objective assessment of urinary incontinence in women: comparison of the one-hour and 24-hour pad tests. *European urology* 45:208-12
- (13) Soroka D (2002) Perineal pad test in evaluating outcome of treatments for female incontinence: a systematic review. *International urogynecology journal* 13:165-75
- (14) Groutz A (2000) Noninvasive outcome measures of urinary incontinence and lower urinary tract symptoms: a multicenter study of micturition diary and pad tests. *The Journal of urology* 164:698-701
- (15) O'Sullivan R (2004) Definition of mild, moderate and severe incontinence on the 24-hour pad test. *BJOG* 111:859-62
- (16) Mouritsen LBGHJ (1989) Comparison of different methods for quantification of urinary leakage in incontinent women. *Neurourol Urodyn* 8:579-87
- (17) Abrams P (2003) The standardisation of terminology in lower urinary tract function: report from the standardisation sub-committee of the International Continence Society. *Urology* 61:37-49
- (18) Jensen JK, Nielsen FR, Jr., and Ostergard DR (1994) The role of patient history in the diagnosis of urinary incontinence. *Obstet Gynecol* 83:904-10
- (19) Lose G, Jorgensen L, and Thunedborg P (1989) 24-hour home pad weighing test versus 1-hour ward test in the assessment of mild stress incontinence. *Acta Obstet Gynecol Scand* 68:211-5
- (20) Karantanis E (2003) The 24-hour pad test in continent women and men: normal values and cyclical alterations. *BJOG* 110:567-71
- (21) Ryhammer AM (1999) Pad testing in incontinent women: a review. *International urogynecology journal* 10:111-5
- (22) Morkved S and Bo K (1999) Prevalence of urinary incontinence during pregnancy and postpartum. *Int Urogynecol J Pelvic Floor Dysfunct* 10:394-8
- (23) Barber MD (2007) Questionnaires for women with pelvic floor disorders. *Int Urogynecol J Pelvic Floor Dysfunct* 18:461-5



# CHAPTER 7

## Summary and conclusions

## 7.1 SUMMARY

### 7.1.1 Introduction

#### 7.1.1.1 Incontinence in pregnancy and after childbirth

Pelvic floor dysfunction in women is a major health problem. Symptoms are protrusion of vaginal tissue, voiding difficulties, urinary incontinence, stool problems and sexual dysfunction. Many of these women may eventually require surgery for pelvic floor dysfunction, especially for prolaps and urinary incontinence. This thesis focuses on urinary incontinence and especially on the etiological aspects of pregnancy and childbirth. In a community survey MacLennan reported a prevalence of all types of self-reported urinary incontinence in women is 35.3%. Urinary incontinence increased after pregnancy according to parity and age. The highest prevalence (51.9%) is reported in women aged 70-74 years<sup>1-4</sup>.

Until now, it is not clear to what extent pregnancy itself or vaginal delivery contributes to the development of urinary incontinence in later life. Following vaginal delivery neuromuscular damage and bladder neck hypermobility, indicating a change in pelvic floor function, has been confirmed<sup>5,6</sup>. Nevertheless in the great majority of women the incontinence has disappeared six months after delivery<sup>7,8</sup>.

#### 7.1.1.2 The urinary continence control system

The control system for urinary continence is a complex network with several components. Anatomical it consist of the urethral support system and sphincteric closing system<sup>9</sup>. This sphincteric closing system yields a closing pressure which may deteriorate due to age<sup>10</sup> and neurological injury<sup>11,12</sup>. Studies on the effect of vaginal birth on the sphincter mechanism reveal decreases in urethral closure pressure as a result of vaginal birth<sup>13,14</sup>.

The urethral support system consists of all the structures extrinsic to the urethra that provide a supportive layer upon which the urethra rests<sup>15</sup>. The following structures can be distinguished:

1. The connective tissue sheath covering the ventral aspects of the urethra and the rhabdosphincter, which may be called ventral urethral fascia, which connects the right and left fasciae of the levator. Contraction of the levator narrows the pre urethral space and an ascending movement of the urethra and the rhabdosphincter.

2. The fasciae of the levator ani muscle, especially the right and left tendineous arch.
3. The strong dorsal structure of the urethra and the rhabdosphincter to the ventral wall of the vagina.

Ultrasound studies have shown that during coughing the inferior abdominal contents are forced to move caudo dorsally (downwards), presumably due to a simultaneous contraction of the diaphragm and abdominal wall muscles. The downward motion of the bladder neck visible in the ultrasound picture means that its surrounding tissues acquire downward momentum. This downward momentum must then be arrested by stretch resistance of the pelvic floor structures.

#### 7.1.1.3 Definition of the problem

*Overstretching of the hiatus urogenitalis during pregnancy and after childbirth.* The ventral fasciae and the fasciae of the levator ani may be separated from the anterior rhabdosphincter. Also, overstretching of the vaginal wall may lead to disruption of the dorsal urethra and rhabdosphincter from the vaginal wall. *As the majority of women has no symptomatic pelvic organ prolaps after vaginal delivery we postulate that both fascia and muscles must have a remarkable accommodation of the level of tissue stretch needed for vaginal birth without major injury of pelvic floor.* During childbirth the urogenital hiatus has to adapt to the passing foetus in a limited time. The increase of the perimeter and/or the straining capacities of the tissue (compliance) of the hiatus urogenitalis might facilitate this process. *There is great scarcity of prospective studies on urinary incontinence during pregnancy and after childbirth.* The wide variation of the reported prevalence and the uncertainty concerning the changes in the continence control system, prompted us to investigate these issues in a prospective longitudinal study in a homogeneous cohort of nulliparous pregnant women. Dynamic serial perineal ultrasound measurements of pelvic floor characteristics may give us a better understanding of the role and the changes in the continence control mechanism, both during pregnancy and after childbirth.

#### 7.1.1.4 Aims of the study

*Assessment of the urethral support system during pregnancy and after childbirth, perineal ultrasonography, compliance and hysteresis.*

By the use of perineal ultrasound and simultaneous abdominal pressure

measurement we were able to measure the displacement and the recovery of the vesical neck in relation to the increase of abdominal pressure during coughing and during Valsalva manoeuvre. In general, deformation under load for tissue is seldomly linear. To quantify such deformation we decomposed deformation into elasticity, approximated by a linear model and hysteresis, estimated by a non linear model. These measurements were made throughout pregnancy and after childbirth resulting in serial measurement of the biomechanical properties of the vesical neck supporting structures during pregnancy and after childbirth.

*Assessment of functional changes of the pelvic floor.* (Functional) injury of pelvic floor, leading to changes of pelvic floor stretch resistance may lead to pelvic floor dysfunction, which includes urinary incontinence. Part of our study therefore was to measure the incidence of this symptom of pelvic floor dysfunction. Women completed questionnaires and visual analogue scales on symptoms of urinary incontinence, moreover 24 hour pad tests were used to objectify urine loss.

In **chapter 2** we assessed the prevalence and the development of urinary incontinence in nulliparous pregnant women, both subjectively and objectively, and we investigated the relation of urinary incontinence with the mobility of the urethro-vesical junction measured by perineal ultrasound.

In **chapter 3** we assessed the prevalence of urinary incontinence after spontaneous vaginal delivery and its relation with changes in the static and dynamic function of the pelvic floor.

In **chapter 4** we compared women with spontaneous and operative vaginal delivery for urinary incontinence data and for pelvic floor characteristics

In **chapter 5** we focused on displacement and recovery of the vesical neck position during pregnancy and after childbirth, especially we discriminated between compliance of the vesical neck supporting structures with and without pelvic floor contraction.

In **chapter 6** we assessed the clinical usefulness of the 24 hour pad test in pregnancy and after childbirth in terms of the relationship between objective urine loss and the self reported symptoms of urinary incontinence.

**Chapter 7** is the summary and the general discussion.

### *7.1.2 Anatomical and functional changes in the lower urinary tract during pregnancy*

The function of the pelvic floor is thought to play an important part in the mechanism of continence in women, and so we investigated the changes in pelvic floor function during pregnancy and its relation to incontinence. We used simultaneous recording of abdominal pressure and the consequent changes in the position of the urethro-vesical junction. The proximal urethra and the anterior vaginal wall are intimately connected and attached to the muscles of the pelvic diaphragm and to the arcus tendineus fasciae pelvis. Support of the urethra comes from both its attachments to the arcus tendineus fasciae pelvis and the resting tone of the muscles of the pelvic diaphragm<sup>16</sup>. Therefore, the mobility of the urethro-vesical junction can be used as an index of the mobility of these pelvic floor structures.

Up to 35% of the women reported urinary incontinence in pregnancy, and 20% of the women had a positive pad test.

The resting angle of the urethro-vesical junction ( $A_0$ ) widened significantly during pregnancy, from 51.5 degrees at 12-16 weeks to 62.0 degrees at term ( $p < 0.001$ ). During coughing there was a significant increasing trend in the displacement/pressure coefficient during pregnancy. For the Valsalva manoeuvre no significant change in the displacement/pressure coefficient was found throughout pregnancy. No relationship was found between measurements of the urethro-vesical junction and the women's perception of urinary incontinence. This applied to all variables studied during coughing as well as during the Valsalva manoeuvre: the angle of the urethro-vesical junction at rest, the angle of the urethro-vesical junction at maximum pressure and the displacement/pressure coefficient. Also, no statistically significant relationship could be demonstrated between these variables and the results of the pad tests.

The results of the present study indicate that the changes in the mobility of the UVJ (a marker for changes of the quality of the connective tissue or muscles of the pelvic floor) are not responsible for the higher prevalence of urine incontinence in pregnancy. Our study focussed on the changes in the extrinsic mechanism of continence, pelvic floor muscle and fascia.



### *7.1.3 Anatomical and functional changes in the lower urinary tract following spontaneous vaginal delivery*

Incontinence in pregnancy is interpreted as a result of interaction between predisposing hereditary factors and uterine pressure upon the bladder, in combination with hormonal effect upon the suspension ligaments of the urethra<sup>17</sup>. Persisting incontinence post partum is mainly the result of changes of the pelvic floor function and anatomy, due to delivery. The (partial) irreversibility of these changes may indicate why stress incontinence appearing for the first time after vaginal delivery has a more serious prognosis than incontinence developing during pregnancy<sup>18</sup>.

After delivery, reported urinary incontinence was reduced 16% and 15% at six weeks and six months after delivery, respectively. Even lower rates were measured by the 24-hour pad test which revealed a decrease to 10% and 5% at 6 weeks and 6 month postpartum, respectively. Our study demonstrates that spontaneous vaginal delivery causes transient as well as long lasting changes in the lower urinary tract. It seems likely that the long lasting changes that we observed six months after delivery will be permanent. Parturition alters urethral support and as a consequence, the position of the bladder neck at rest becomes permanently descended. The changes in pelvic floor reaction to coughing, as measured by an increase of the displacement/pressure coefficient, indicates reduced pelvic floor stiffness due to coughing. This finding adds to the effect of pregnancy, where we observed already a significant decrease in pelvic floor stiffness during coughing<sup>19</sup>. As in pregnancy we did not find any relation between incontinence measurements, urethro-vesical junction measurements and obstetric variables.

It is concluded that pregnancy and spontaneous vaginal delivery significantly permanently alter the static condition of bladder neck descent. The dynamics of the pelvic floor are affected as well. This effect is temporary for Valsalva and permanent for coughing. Urinary incontinence, quite common during pregnancy, disappears post partum in most women. No evidence was found for a significant association between pelvic floor function measurement and urinary incontinence, nor at 6 weeks, nor at 6 months postpartum. As the onset of symptoms of urinary incontinence later in life is thought to be due to the combined effect of occult trauma during pregnancy and delivery and the progression of neuropathy during lifetime<sup>5,20</sup> the persistent anatomical and functional changes in the lower urinary tract that we found, could play an important role in the aetiology of urinary stress incontinence later in life.

#### *7.1.4 Pelvic floor characteristics after spontaneous and operative vaginal delivery. Serial studies prior to labor up to six months postpartum*

In this study post partum (hyper) mobility is not influenced by the type of delivery, but by the ante partum mobility and therefore seems to be determined by (intrinsic) patient characteristics. The present data showed no significant differences for urinary incontinence between the group of women who delivered spontaneously and the group who had an operative vaginal delivery. Women with operative and spontaneous vaginal delivery have comparable ante partum and postpartum pelvic floor characteristics. Therefore, if these changes are predictors of incontinence later in lifetime, in both groups women are at the same risk to develop incontinence later.

Birth parameters, such as duration of labor, birth weight and head circumference did not correlate with pelvic floor characteristics as well. The finding that these extrinsic factors do not influence pelvic floor characteristics is in agreement with the conclusions of others who emphasize the influence of intrinsic factors, such as genetically determined characteristics of the collagen tissue<sup>21-23</sup>.

In conclusion we found that pelvic floor characteristics change as a result of vaginal delivery in nulliparous women. These changes occur after spontaneous as well as after operative vaginal delivery. As to these changes we found no significant differences between the spontaneously and the operatively delivered group. Also for urinary incontinence, both measured subjectively and objectively, we found no significant differences between the two groups of women. Extrinsic factors predicting pelvic floor dysfunction, such as birth related factors, were not identified. As we found that ante partum compliance is significantly correlated with post partum values, intrinsic factors, such as collagen characteristics, could be more important.

#### *7.1.5 Displacement and recovery of the vesical neck position during pregnancy and after childbirth*

We focussed on the biomechanical properties of the vesical neck supporting structures during pregnancy and after childbirth. Generally, tissue distensibility is determined not only by the elasticity of the constituent muscle and connective fibers itself but also by the geometrical arrangement of those fibers. Two parameters are in use to describe the mechanical properties of tissue: Young's modulus, or its reciprocal (the compliance), to quantify the pure elastic behaviour of tissue in which the geometrical structure of the fibers remain unaltered, and hysteresis: the failure

of tissue to follow the same course during relaxation as it did during distension. The latter parameter is thought to be the result of shifts in the geometrical structure of the fibers with respect to each other, and can be interpreted as a form of internal friction within the tissue.

Also, inertial effects involved may effect the return of the tissues to a normal position.

In our study the increase in hysteresis seems to be permanent. Since hysteresis, the phenomenon that tissue does not follow the same course during distention as during relaxation, depends on shifts of the constituent fibers with respect to each other, an increase in hysteresis means that the geometrical structure becomes looser. Both compliance and hysteresis contribute to the stiffness of the pelvic floor and our data suggest that a decrease in stiffness of the pelvic floor after child birth is mainly due to a more loosely coupling of the various fibers in the pelvic floor tissue to each other. Reduced fascial strength and decreased muscle tone tend to make the pelvic floor more flaccid so that the necessary back pressure cannot be produced leading to hypermobility of the Urethro Vesical Junction, significantly related with genuine urinary stress incontinence<sup>24</sup>.

Hysteresis showed an increase after child birth at least persisting until six months post partum, showing that delivery may stretch and or load beyond the physiological properties of the pelvic floor tissue and in this way may lead to irreversible changes in tissue properties which play an important role in the urethral support continence mechanism.

#### ***7.1.6 The diagnostic strength of the 24-hour pad test for self reported symptoms of urinary incontinence in pregnancy and after childbirth***

In our study during pregnancy and after childbirth pad test results had only limited diagnostic value for self reporting of incontinence. Pregnancy seems to modulate this perception in such a way that it cannot be measured by pad test. As pad testing did not show to have high sensitivity and specificity for self reported urinary incontinence in pregnancy and after childbirth there remains confusion about the accurate diagnosis. This becomes important in deciding on management options such as offering preventive physiotherapy in selected cases or strategies which influence the mode of delivery.

From our study we conclude that pad testing may measure fluid loss over a certain period, but does not quantify self reported symptoms of urinary incontinence. Both

measurements may be of interest, but cannot replace each other. Stressing of the pelvic floor by pregnancy and childbirth modulates the sensation of urinary leakage as measured by the pad test in such a way that women in this state do report symptoms of urinary incontinence more frequently than nulliparous premenopausal women do.

When using these figures to establish the role of pregnancy and childbirth in the risks of the development of urinary incontinence one should realise this modulation effect.

## 7.2 CONCLUSIONS

### *7.2.1 Assessment of the urethral support system during pregnancy and after childbirth, perineal ultrasonography, compliance and hysteresis*

In our study on pelvic floor parameters during pregnancy we were able to measure adaptation effects. The resting angle, a marker for perineal descent, is widening, already at 12-16 weeks of pregnancy. We also measured an increase of the compliance both during Valsalva and during coughing. In conclusion two main elements of adaptation can be distinguished. First of all the lengthening of the tissue sling surrounding the vagina, the birth canal, and secondly more distensibility of this sling.

We described an adaptive effect of pregnancy on the capacity of the birth canal in such a way that vaginal birth can be realized without either serious tearing of the hammock tissue or serious damage to the passing fetal head.

These changes were found at least until 6 months post partum and must therefore be considered as definitively. First of all the widening of the resting angle, marking the lowering of the central point of the perineum, at which the pelvic floor tissue inserts from right, left anterior and posterior. This lowering will lead to changes in supportive properties of the pelvic floor because of the longer distance between the urethral back side and the supportive structures, which may even lead to prolaps of the vaginal anterior wall.

Moreover the dynamic properties of the tissue changes as can be concluded from the increased compliance. Hysteresis, a measurement of internal tissue friction due to rearrangement of tissue under load, is not affected by pregnancy. Tissue load keeps up with physiological margins. But after delivery a remarkable increase in

hysteresis occurred, remaining at least until 6 months. If considered as a persisting phenomenon the dynamics of the urethral support system are affected definitively. We concluded that there is a change in the supportive structures of the urethra during pregnancy and after childbirth. These changes were measured both after spontaneous and after operative vaginal delivery. Because of the limited numbers caution must be taken to conclude about the preferable way of delivery. Caesarean Sectio numbers were too small and indications were heterogeneous, we did not report about these data. In this study post partum (hyper) mobility is not influenced by the type of delivery, but by the ante partum mobility and therefore seems to be determined by (intrinsic) patient characteristics.

### *7.2.2 Assessment of functional changes of the pelvic floor*

During pregnancy we found an increase of women complaining about symptoms of urinary incontinence, already decreasing as early as 6 weeks post partum. We did not find any relation between incontinence parameters and pelvic floor parameters. Either there is no such relationship, or this relationship is overruled by other systems contributing to urinary continence in women.

In this thesis we focused on the extrinsic urinary continence system. The intrinsic urethral sphincteric closing system is also known to contribute to continence in women. This intrinsic system physiologically declines with age. This means that continence in elder women is more and more depending on the extrinsic urethral supporting system. Occult damage to the urethral support system may become recognizable only in later life.

Incontinence in pregnancy may be more dependent on the sphincteric closing system as already early found by van Geelen en Iosif. Our research was pointed to the changes in the urethral supportive system in pregnancy and after childbirth. This may explain why we did not find a relation between our pelvic floor parameters and incontinence measurements, confounded by the role of the intrinsic sphincteric system.

As patients and doctors do feel that incontinence in pregnancy needs preventive strategy, an objective measurement is needed. We tested the diagnostic strength of the 24-hour pad test for self reported symptoms of urinary incontinence in pregnancy and after childbirth. The 24 hour pad test did not meet the criteria for a reliable clinical test. In many ways it did not distinguish between women with and without reporting of urinary incontinence.

The results of our study raise the question whether or not ultrasound measurement reveals prognostic data on which selection of patients for preventive strategies is thoughtful. As we found that post partum change in pelvic floor function is only related with ante partum values, and if we think that post partum pelvic floor parameters are prognostic for GSI later in life, ultrasound could be helpful. This is only speculative and should be the issue of future investigation.

## REFERENCE LIST

- (1) MacLennan AH, Taylor AW, Wilson DH, Wilson D. The prevalence of pelvic floor disorders and their relationship to gender, age, parity and mode of delivery. *BJOG* 2000; 107(12):1460-1470.
- (2) Peyrat L. Prevalence and risk factors of urinary incontinence in young and middle-aged women. *BJU international* 2002; 89(1):61-66.
- (3) Lukacz ES, Lawrence JM, Contreras R, Nager CW, Luber KM. Parity, mode of delivery, and pelvic floor disorders. *Obstet Gynecol* 2006; 107(6):1253-1260.
- (4) Chiarelli P, Brown WJ. Leaking urine in Australian women: prevalence and associated conditions. *Women Health* 1999; 29(1):1-13.
- (5) Sultan AH, Kamm MA, Hudson CN. Pudendal nerve damage during labour: prospective study before and after childbirth. *Br J Obstet Gynaecol* 1994; 101(1):22-28.
- (6) Meyer S, Schreyer A, De Grandi P, Hohlfeld P. The effects of birth on urinary continence mechanisms and other pelvic-floor characteristics. *Obstet Gynecol* 1998; 92(4 Pt 1):613-618.
- (7) Viktrup L. The risk of stress incontinence 5 years after first delivery. *American journal of obstetrics and gynecology* 2001; 185(1):82-87.
- (8) Thorp JM, Jr., Norton PA, Wall LL, Kuller JA, Eucker B, Wells E. Urinary incontinence in pregnancy and the puerperium: a prospective study. *Am J Obstet Gynecol* 1999; 181(2):266-273.
- (9) Ashton-Miller JA, Howard D, DeLancey JO. The functional anatomy of the female pelvic floor and stress continence control system. *Scand J Urol Nephrol Suppl* 2001;(207):1-7.
- (10) Rud T, Andersson KE, Asmussen M, Hunting A, Ulmsten U. Factors maintaining the intraurethral pressure in women. *Invest Urol* 1980; 17(4):343-347.

- (11) Hilton P, Stanton SL. Urethral pressure measurement by microtransducer: the results in symptom-free women and in those with genuine stress incontinence. *Br J Obstet Gynaecol* 1983; 90(10):919-933.
- (12) Snooks SJ, Badenoch DF, Tiptaft RC, Swash M. Perineal nerve damage in genuine stress urinary incontinence. An electrophysiological study. *Br J Urol* 1985; 57(4):422-426.
- (13) Iosif S, Ulmsten U. Comparative urodynamic studies of continent and stress incontinent women in pregnancy and in the puerperium. *Am J Obstet Gynecol* 1981; 140(6):645-650.
- (14) van Geelen JM, Lemmens WA, Eskes TK, Martin CB, Jr. The urethral pressure profile in pregnancy and after delivery in healthy nulliparous women. *Am J Obstet Gynecol* 1982; 144(6):636-649.
- (15) Fritsch H, Pinggera GM, Lienemann A, Mitterberger M, Bartsch G, Strasser H. What are the supportive structures of the female urethra? *Neurourol Urodyn* 2006; 25(2):128-134.
- (16) DeLancey JO. Correlative study of paraurethral anatomy. *Obstet Gynecol* 1986; 68(1):91-97.
- (17) Iosif CS, Ingemarsson I. Prevalence of stress incontinence among women delivered by elective cesarian section. *Int J Gynaecol Obstet* 1982; 20(2):87-89.
- (18) Viktrup L, Lose G, Rolff M, Barfoed K. The symptom of stress incontinence caused by pregnancy or delivery in primiparas. *Obstet Gynecol* 1992; 79(6):945-949.
- (19) Wijma J, Weis Potters AE, de Wolf BT, Tinga DJ, Aarnoudse JG. Anatomical and functional changes in the lower urinary tract during pregnancy. *BJOG* 2001; 108(7):726-732.
- (20) Snooks SJ, Swash M, Mathers SE, Henry MM. Effect of vaginal delivery on the pelvic floor: a 5-year follow-up. *Br J Surg* 1990; 77(12):1358-1360.
- (21) Keane DP, Sims TJ, Abrams P, Bailey AJ. Analysis of collagen status in premenopausal nulliparous women with genuine stress incontinence. *Br J Obstet Gynaecol* 1997; 104(9):994-998.
- (22) Dietz HP, Hansell NK, Grace ME, Eldridge AM, Clarke B, Martin NG. Bladder neck mobility is a heritable trait. *BJOG* 2005; 112(3):334-339.
- (23) King JK, Freeman RM. Is antenatal bladder neck mobility a risk factor for postpartum stress incontinence? *Br J Obstet Gynaecol* 1998; 105(12):1300-1307.
- (24) DeLancey JO. Structural support of the urethra as it relates to stress urinary incontinence: the hammock hypothesis. *Am J Obstet Gynecol* 1994; 170(6):1713-1720.



# CHAPTER 8

**Samenvatting en conclusies  
(Summary and conclusions in Dutch)**



## 8.1 SAMENVATTING EN CONCLUSIES

### 8.1.1 Inleiding

#### 8.1.1.1 Incontinentie tijdens de zwangerschap en na de baring

Bekkenbodem disfunctie vormt een significant gezondheidsprobleem. Symptomen zijn verzakking, mictie- en defaecatieproblematiek en seksuele disfunctie. Dit proefschrift beperkt zich tot incontinentie en speciaal tot de etiologische aspecten ervan, zwangerschap en baring. Tijdens de zwangerschap komt incontinentie bij 35% van de vrouwen voor. Bij toename van pariteit en leeftijd neemt dit toe tot ruim 50% bij vrouwen van 70 jaar en ouder. Er zijn veel retrospectieve onderzoeken die een relatie tussen de baring en incontinentie aannemelijk maken. Er zijn echter maar weinig prospectieve studies over dit onderwerp. Bovendien is de rol van de zwangerschap in het ontstaan van incontinentie onduidelijk. Tijdens de zwangerschap speelt de veranderde hormoonhuishouding naast veranderde anatomische verhoudingen een rol. Door de zwangerschap uitgelokte veranderingen in het bindweefsel zouden ook op langere termijn consequenties kunnen hebben. Van de partus wordt gedacht dat deze zowel tot beschadiging van het bindweefsel als tot neuromusculaire beschadigingen kan leiden, en daarmee verantwoordelijk moet worden gehouden voor blijvende beschadiging van het steun- en fixatie weefsel van de blaashals. Dit is een licht voorstelbaar concept, anderzijds is ook bekend dat de meerderheid van de post partum voorkomende incontinentieklachten spontaan afnemen. Hoewel dit een aannemelijk concept lijkt, is echter ook bekend dat de meerderheid van de post partum voorkomende incontinentieklachten spontaan afneemt.

#### 8.1.1.2 Het urine continentie systeem

Het continentiesysteem is een complex systeem, bestaande uit het intrinsieke urethrale afsluitsysteem en het extrinsieke "urethral support " systeem.

Het intrinsieke systeem bestaat uit glad en dwarsgestreept spierweefsel en de vasculaire componenten van de submucosa. Dit intrinsieke systeem verliest met toename van de leeftijd aan functionaliteit door apoptosis en neuromusculaire schade. Ook na een vaginale partus is een afname van urethrale sluitdruk vastgesteld. Het lange termijn effect is onduidelijk.

Het extrinsieke systeem bestaat uit een aantal weefsellagen waar de blaashals op rust bestaande uit bindweefsel schotten, fascia van de spier en spierweefsel

met dwarse oriëntatie, onder de urethra en blaas door aan beide zijden lateraal aanhechtend op het bekken. Tijdens hoesten worden buikdrukken van 150 cm H<sub>2</sub>O gegenereerd. Bij echografie is zichtbaar dat de buikinhoud naar caudaal beweegt. De blaashals beweegt mee, maar wordt gestopt door weefselweerstand tengevolge van de opgetreden weefselrek. De urethra wordt ter plaatse van de urethro vesicale overgang dichtgedrukt tussen enerzijds de caudaal bewegende buikinhoud en anderzijds de craniaal gerichte kracht tengevolge van de weefselrek van het ondersteunend weefsel.

#### 8.1.1.3 De probleemstelling

Overrekking van de hiatus urogenitalis tijdens zwangerschap en na de baring. Terwijl de diameter van de hiatus voor de eerste zangerschap 2.5 centimeter bedraagt is de diameter van de passerende foetale schedel het viervoud, 9 centimeter. Daarvoor is een uitzettingsratio van 3.6 noodzakelijk, dit is 217% van de maximale uitzettingsratio van de samenstellende weefsels (1,5). Uit eerder onderzoek blijkt dat er bij beschadiging van de hiatus urogenitalis met name voor achterwaartse beschadiging op te treden. Daarmee desintegreert de spiergroep van de levatoren en daarmee de dakpansgewijze klepwerking van de hiatus urogenitalis, leidend tot verminderde ondersteuning van de in de buikholte gelegen organen, zoals de blaas en de blaashals. Daarmee ontstaat de kans op incontinentie en prolaps.

Omdat een groot deel van de vrouwen deze klachten niet heeft moet er tijdens de zwangerschap en/of baring een mechanisme zijn waardoor weefselrek in staat is om zich zodanig aan te passen dat zwangerschap en partus kunnen plaatsvinden zonder ernstige weefselschade.

#### 8.1.1.4 Doel van de studie

*Het vastleggen van de functionele anatomie van het ondersteunend weefsel van de blaashals tijdens de zwangerschap en na de baring, zowel in rust als onder belasting.*

De positie in rust en de bewegingen van de blaashals (gemeten door middel van perineale echografie) worden gerelateerd aan buikdrukverhoging tijdens hoesten en tijdens persen. Bij persen is de buikdruk verhoogd en de bekkenbodem ontspannen en wordt de rek van de bekkenbodem vooral bepaald door de rekbaarheid van de samenstellende delen, vereenvoudigd benaderd als de collageen eigenschap.

Tijdens hoesten is de buikdruk verhoogd en worden de bekkenbodemspieren in meer of mindere mate aangespannen. Daardoor kunnen verschillen in rekbaarheid

gemeten worden. Onderzocht wordt de verandering van de positie in rust en de verandering in rekbaarheid van het ondersteunend weefsel van de blaashals. Tenslotte de relatie ervan met de zwangerschap, de baring en andere obstetrische parameters.

*Het vastleggen van de functionele veranderingen tijdens de zwangerschap en na de baring.* Door seriële vragenlijsten en 24 uur luiertesten wordt inzicht verkregen in de mate van voorkomen en de ernst van het klinische symptoom, incontinentie voor urine. Onderzocht wordt of er veranderingen optreden in de mate van voorkomen en de ernst in relatie tot zwangerschap, baring en andere obstetrische parameters. Tenslotte wordt onderzocht of veranderingen in de rustpositie en de rekbaarheid van het ondersteunend weefsel van de blaashals leiden tot veranderingen in de symptomatologie, de mate en de ernst van het voorkomen van urine incontinentie.

#### ***8.1.2 Anatomische en functionele veranderingen van de lagere urinewegen tijdens de zwangerschap***

De bekkenbodem geeft steun aan de blaashals en heeft een belangrijke functie in het continentie mechanisme van de vrouw. Wij bestudeerden de veranderingen daarvan tijdens de zwangerschap en de relatie daarvan met het optreden van incontinentie. Door het meten van veranderingen in de buikdruk en de dientengevolge optredende veranderingen van de positie van de blaashals kan een uitspraak gedaan worden over de mechanische eigenschappen van de weefsels waarop de blaashals rust, de bekkenbodem. De belangrijkste componenten zijn het steun en fixatie weefsel en de spieren waarop de blaashals rust.

Incontinentie komt tijdens de zwangerschap frequent voor. Van de zwangeren geeft 35% aan incontinent te zijn, terwijl bij 20% een positieve luiertest wordt gemeten. Dit is een significante toename in vergelijking met leeftijdsgenoten die nooit zwanger waren. Reeds vroeg in de zwangerschap treedt in rust descensus van de bekkenbodem op in relatie tot een vast meetpunt, de onderrand van de symfyse. Tijdens hoesten neemt de mobiliteit toe, terwijl bij persen deze toename niet gevonden wordt.

Er is geen relatie gemeten tussen incontinentie en de mate van descensus en de mate van elasticiteit van de bekkenbodem. Dit geldt zowel voor gepercipieerde als gemeten incontinentie.

### *8.1.3 Anatomische en functionele veranderingen van de lagere urinewegen na de spontane vaginale baring*

Incontinentie post partum wordt toegeschreven aan functionele en anatomische veranderingen van de bekkenbodem tengevolge van de mechanische belasting tijdens de baring. In vergelijking met het voorkomen tijdens de zwangerschap neemt post partum de prevalentie duidelijk af. Na 6 maanden geeft nog 15% aan incontinent te zijn, terwijl bij 5% nog een positieve luiertest wordt gemeten. veranderd. Zowel de statische als de dynamische eigenschappen zijn na de spontane vaginale baring blijvend veranderd, dat wil zeggen na een periode van 6 maand na de baring. Ondersteuning van de blaashals is daarmee blijvend veranderd.

Ook na de baring zijn de veranderingen in het steun en fixatie mechanisme van de blaashals niet gerelateerd aan het voorkomen van incontinentie. Niettemin kunnen de veranderingen op latere leeftijd van grote betekenis blijken wanneer het intrinsieke continentie systeem fysiologisch aan waarde inboet.

### *8.1.4 Anatomische en functionele veranderingen van de lagere urinewegen na spontane en geassisteerde (vacuüm/forceps) vaginale baring*

In dit hoofdstuk worden de uitkomsten van incontinentieklachten en mobiliteit van de bekkenbodem na spontane en geassisteerde vaginale baringen vergeleken. Zowel tijdens de zwangerschap als na de baring komt incontinentie in beide groepen vergelijkbaar voor. Dit geldt evenzeer voor de bekkenbodem parameters, de mobiliteit van de urethro vesicale overgang. Niet het type baring is een voorspeller van de veranderingen in de bekkenbodem parameters, maar wel is er een significante relatie met de uitgangswaarde. Met andere woorden, het zijn patiëntgebonden eigenschappen. Dit past bij de opkomende opvatting dat genetische eigenschappen van het collageen van doorslaggevend belang zijn voor het al dan niet ontstaan hypermobiliteit van de bekkenbodem. Deze hypermobiliteit is op latere leeftijd vervolgens sterk gecorreleerd aan incontinentie.

### *8.1.5 Verplaatsing en herstel van de positie van de blaashals tijdens de zwangerschap en na de partus*

In dit hoofdstuk wordt nader ingegaan op de biofysische eigenschappen van structuren die de blaashals ondersteunen. De bewegingen van de blaashals worden modelmatig ontleed. In vergelijkbare studies wordt de elasticiteit gemeten. Naast elasticiteit van weefsel is ook de hysteresis bepalend voor het biofysische gedrag. Door interne geometrische veranderingen ontstaat frictie tussen de samenstellende weefselstructuren, collageen- en spiervezels. Deze frictie verbruikt energie en heeft een niet lineair bewegingspatroon tot gevolg, weefseltraagheid. Zowel de elasticiteit als deze weefseltraagheid beïnvloeden de ondersteuning van de blaashals en kunnen daardoor een belangrijke rol spelen bij het ontstaan van incontinentie.

In de zwangerschap treedt een toename van de elasticiteit van de weefsels op. Zowel bij hoesten als bij Valsalva. Tijdens de zwangerschap blijft de hysteresis onveranderd. Post partum is er een significante toename van hysteresis, persisterend tot 6 maanden post partum, terwijl de elasticiteit grotendeels normaliseert. Voor Valsalva geheel, bij hoesten nagenoeg. De persisterende verandering van hysteresis is het gevolg van een verandering van de geometrische organisatie van het weefsel, een teken van overrekking van het weefsel tijdens de baring.

### *8.1.6 De diagnostische waarde van de 24 uur padtest voor de klacht urine incontinentie in de zwangerschap en na de baring*

Uit onze studie blijkt maar een beperkte waarde van de pad test voor de klacht urine incontinentie. Ook voor het objectiveren van subjectieve ernst van incontinentie heeft de pad test weinig waarde. Juist nu het obstetrisch handelen beïnvloed dreigt te worden door incontinentieklachten tijdens de zwangerschap is het stellen van een accurate diagnose belangrijk. Ook voor interventie(studies) door fysiotherapie is een eenduidig diagnostisch proces van groot belang.

Uit onze studie concluderen wij dat de pad test niet gebruikt kan worden om de subjectieve klacht incontinentie te kwantificeren. Voor de kliniek is de pad test geen aanwinst. Voor studies naar incontinentie en therapeutische interventies blijft de pad test een plaats houden. De lekkage in de pad test was niet anders dan bij niet zwangeren, terwijl het aantal vrouwen dat zichzelf als incontinent aanmerkt significant hoger is. Blijkbaar moduleert zwangerschap de sensatie van incontinentie op een significante wijze.

## 8.2 CONCLUSIES

### *8.2.1 Het vastleggen van de functionele anatomie van het ondersteunend weefsel van de blaas urethra overgang tijdens de zwangerschap en na de baring, zowel in rust als onder belasting*

In deze studie naar de functionele anatomie van het ondersteunend weefsel van de blaas urethra overgang werd tijdens de zwangerschap zowel descensus van de bekkenbodem als een toename van de elasticiteit gemeten. Deze toegenomen descensus en elasticiteit wordt reeds vroeg (12-16<sup>e</sup> week) in de zwangerschap gemeten. Zowel de initiële omvang van de hiatus urogenitalis als capaciteit tengevolge van rek van het samenstellende weefsel verhogen de kans op een geslaagde passage van de foetale schedel door het geboortekanaal zonder significante beschadiging ervan.

Naast een partieel voorbijgaande toegenomen elasticiteit is er wel een persisterende verandering van weefseleigenschappen meetbaar, namelijk een toegenomen hysteresis. Dit duidt erop dat er wel degelijk weefselschade optreedt, namelijk een veranderde interne weefsel architectuur.

In onze studie groep is er voor de functionele anatomische veranderingen geen verschil aantoonbaar tussen vrouwen die spontaan bevielen dan wel per vaginale kunstverlossing. Echter, de groepsgrootte is niet toereikend om een eenduidige conclusie te trekken.

In deze studie blijken de veranderingen in de functionele anatomie niet zozeer bepaald door het type baring als wel door de initiële waarde. Dit pleit voor intrinsieke weefseleigenschappen die bepalend zijn voor de uitkomst, aansluitend bij de huidige idee dat genetische eigenschappen het meest bepalend zijn voor de kans op bekkenbodem disfunctie.

### *8.2.2 Het vastleggen van de functionele veranderingen tijdens de zwangerschap en na de baring*

Het aantal vrouwen met incontinentieklachten neemt al vroeg in de zwangerschap toe. Post partum is er na 6 weken weer een duidelijke afname. Zowel in de zwangerschap als daarna is er geen relatie tussen bekkenbodem parameters en incontinentie, dit geldt voor gerapporteerde en gemeten incontinentie. Ofwel er is geen relatie, ofwel de relatie wordt overstemd door een andere meer significant fenomeen.

In deze studie is het focus gericht op het zogenaamde extrinsieke systeem. Het intrinsieke systeem heeft ook een belangrijke bijdrage aan het continentie systeem. De bijdrage van dit intrinsieke systeem neemt fysiologisch af met de leeftijd. Dit zou kunnen betekenen dat oudere vrouwen meer en meer afhankelijk worden van hun extrinsieke systeem. Occulte beschadiging (tijdens zwangerschap en baring) zou daardoor pas op latere leeftijd zichtbaar worden.

Incontinentie tijdens de zwangerschap en na de baring zou meer afhankelijk kunnen zijn van het tijdelijk verminderd functionerende intrinsieke systeem. Dit fenomeen zou de relatie met de veranderde functionele anatomie kunnen beïnvloeden.

Omdat zowel zwangeren als artsen interventie keuzes overwegen op basis van het fenomeen incontinentie is er groeiende behoefte aan een objectief criterium voor het vaststellen van urine incontinentie. Wij testten de diagnostische waarde van de 24 uren luiertest voor subjectief gerapporteerde incontinentie, zowel voor de ernst als voor het voorkomen op zich. De luiertest voldoet niet aan de criteria voor een betrouwbare klinische test. Voor interventiestudies blijft er een plaats, voor klinische doeleinden voldoet een vragenlijst.

De vraag is of echografisch onderzoek van de bekkenbodem in de zwangerschap of na de baring naast etiologische beschouwingen ook relevante prognostische informatie voor de individuele patiënt kan opleveren. Op basis van de vaststelling dat ante partum data bepalend zijn voor post partum data, en op basis van de veronderstelling dat hypermobilititeit een belangrijke etiologische factor voor het ontstaan van incontinentie is, zou echografisch onderzoek behulpzaam kunnen zijn bij het traceren van individuele risico's. Dit is echter speculatief en zal eerst onderwerp van nader onderzoek moeten zijn.

## NAWOORD

Dit proefschrift heeft lang op zich laten wachten. Dat lag niet aan anderen, dat was het gevolg van kansen en keuzes.

Het doen van wetenschappelijk onderzoek was voor mij uitdagend en leerzaam. In de praktijk bleek het succes afhankelijk van de inzet van vele mensen. Mijn bewondering, met name voor het geduld en het vermogen om mij te inspireren. Het noemen van namen doet anderen tekort. Ik ben ieder zeer erkentelijk voor de inzet. Dit geldt voor de collega's van de beide klinieken, maar ook voor al die geduldige patiënten die de moeite hebben genomen om bij herhaling speciaal voor het onderzoek naar de kliniek te komen.

Naast het wetenschappelijk onderzoek bood het leven steeds vele kansen die de moeite meer dan waard waren. Het werk als gynaecoloog in het Martiniziekenhuis biedt me de mogelijkheid om cliënten een waardevolle dienst te bewijzen. Overheid en verzekeraars, zij praten over de polis en niet over de inhoud, lijken ons anders te laten geloven. Het lijkt er op dat wij onze energie vooral aan hen dreigen te verliezen. De waarde van onze diensten wordt door onze cliënten zelden of nooit in twijfel getrokken. Deze overtuiging is de inspiratiebron van mijn belangstelling voor de inhoud van de zorg en de organisatie ervan.

Investeren in al deze facetten van het werk of in privé lijkt een moeizame keuze die ik zelden zo heb ervaren. Dat laatste ervaar ik als een kans die me wordt geboden, als dank daarvoor dan toch enkele namen,

Nienke, Jan en Marian



## **CURRICULUM VITAE**

Jacobus Wijma was born in Leeuwarden, the Netherlands, on August 8<sup>th</sup> 1958. He attended primary school and secondary school in Leeuwarden and Groningen and completed his medical studies in 1984 at the University of Groningen. He specialized in Obstetrics and Gynaecology from 1988 to 1993 at the University Medical Centre Groningen and the Martini Hospital Groningen. Since 1993 he is working as a member of staff at the Martini Hospital Groningen.

The research for this thesis was done since 1993 at the University Medical Centre and the Martini Hospital. Apart from this he participated in many committees both in the Martini Hospital and in the NVOG, the Dutch association of gynaecologists.

He was president of the medical board from the Martini Hospital from 1999-2002. He was one of the founders of the "Coöperatie van Vrijgevestigd Medisch Specialisten van het Martiniziekenhuis". Since 2002 he participated in several national committees concerning the development and introduction of the DBC, the new finance system of the health care in the Netherlands. He is addicted to sailing and has two wonderful children Jan (1987) en Marian (1989) and is married to his beloved Nienke Idsinga.

# **Korte Nederlandse samenvatting bij het proefschrift van Jacobus Wijma**

## **The urethral support system during pregnancy and after childbirth**

**10 december 2007**

### **INLEIDING**

Tijdens de zwangerschap komt ongewild urineverlies bij 35% van de vrouwen voor. Bij toename van het aantal bevallingen en van de leeftijd stijgt dit percentage tot 50%.

Terwijl de diameter van het geboortekanaal voor de eerste zwangerschap 2.5 centimeter bedraagt, is de diameter van het passerende kindshoofd het viervoud hiervan (9 centimeter). Dit vereist dus aanpassingen van het geboortekanaal.

### **DOEL VAN DE STUDIE**

Het meten van aanpassingen van het geboortekanaal die nodig zijn om overrek en afscheuren van weefsel te voorkomen en het bestuderen van de relatie tussen weefselaanpassingen en incontinentie voor urine.

### **RESULTATEN**

Al vroeg in de zwangerschap wordt een lagere positie van de bekkenbodem gemeten, resulterend in een grotere diameter van het geboortekanaal. Na de geboorte blijkt de rekbaarheid te zijn toegenomen. Zowel de omvang van het geboortekanaal als de capaciteit tengevolge van verhoogde rekbaarheid verhogen de kans op een geslaagde passage van het kind zonder ernstige beschadiging van kind en geboortekanaal.

Toch is er gemiddeld een effect meetbaar dat duidt op blijvende verandering van de interne weefsel architectuur, wijzend op overrekking.

De mate waarin de weefselaanpassingen optreden lijken patiënt gerelateerd, en niet afhankelijk van het type vaginale baring.

Het aantal vrouwen met incontinentieklachten neemt al vroeg in de zwangerschap toe. Na de bevalling is er weer een duidelijke afname. Zowel in de zwangerschap als daarna is er geen relatie tussen bekkenbodemp parameters en incontinentie.

## **CONCLUSIE**

Tijdens de zwangerschap en rondom de baring treden weefselaanpassingen op die een geslaagde vaginale baring mogelijk maken. Desondanks treedt er gemiddeld overrekking van de bekkenbodemp op. De mate waarin deze aanpassing plaatsvindt, blijkt vooral patiëntgebonden.